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An Address.¹

THE RELATION OF THE BRITISH MEDICAL ASSOCIATION TO STATECRAFT.

By J. E. FERGUSON STEWART, M.B., Ch.M. (Glasgow),
Retiring President of the Western Australian
Branch of the British Medical
Association.

It is increasingly evident every year that governments are giving more heed towards the welfare of the communities governed. This is evidenced in recent years by the establishment of Ministries of Health and Departments of Public Health to which powers have been granted to supervise the welfare and particularly the health of the community.

I doubt if as a body we can claim much credit in the initiation of these much needed institutions and the reforms that have been brought about, but no doubt the efforts of individual members of our profession must be given some credit.

I have thought that as a body perhaps we do not make our influence sufficiently felt in the com-

munities in which we live, and perhaps we have a good excuse in that such matters, being under the control of political bodies, are liable to become matters of party contention in which we have no desire to embroil ourselves.

It is a question, however, if as a body we could not from time to time make suggestions to some extent on questions that affect the welfare of the community and there seems to be a section of the profession in the old country led by Sir Arbuthnot Lane decrying that aloofness from matters of public interest which has characterized our Association.

Let me hasten to add that I do not wish for a moment to suggest any alteration to our rule regarding press publicity by individuals, but I can well believe that a carefully considered published opinion from the Council of the Branch of the British Medical Association or from a general meeting on some matter which is exercising the public mind as well as that of legislators, might be very welcome, if it were known that such an opinion could be obtained. It may be argued that the legislature can always get the opinion of its departmental medical officers on these matters, but I submit that in many subjects these officers are not so well situated to express opinions as are the men moving

¹ Delivered at the Annual Meeting of the Western Australian Branch of the British Medical Association on March 21, 1926.

in daily contact with the home lives of all classes of the community.

That there is a desire to "rope in" the general practitioner's services in regard to public health was clearly shown by the Commission appointed by the Commonwealth Government which recently took evidence in this as well as the other States, a desire for statistics in regard to the incidence of morbidity of all kinds and not only the statistics of mortality such as can be gleaned from the registrar of births.

I feel myself that such statistics would be valuable in many ways to legislative bodies. Take as an instance the number of minor injuries and maimings caused by the promiscuous sale of firearms. We get numbers of these which have more or less serious results, but none are recorded anywhere fully unless fatal results ensue. Some are published in the press, but this is as we know a very inaccurate source of information in a large percentage of cases and of little value for statistics besides being a laborious method for the collators of statistics.

Other matters on which our opinion might well carry weight are those of:

(i.) Segregation and sterilization of the mentally defective.

(ii.) Advice as to methods to insure more cleanliness in individuals as an essential to freedom from disease in the community.

(iii.) The advisability or otherwise of the admission of other races than our own and of other nationalities to Australia.

On these questions I consider that as a body we have some right to be heard and that it is our duty to inform our governing bodies of the more remote effects which their legislation may have.

None of these matters are properly speaking "party" questions, but are questions of statesmanship and at the risk of being rebuffed I believe we should revise our policy of silence.

From the present arguments going on in the press regarding the immigration of southern Europeans our politicians are only interested in whether the newcomers will lower wages or produce increased wealth for the community. No doubt this is an important aspect, but it is also what I would term the mercenary and shortsighted aspect. What is far more important is "what influence is this immigration going to have on the future of the Australian nation?" Are these people physically and mentally likely to raise or lower the Anglo-Saxon standard? What percentage can we assimilate without risking Australia becoming a polyglot collection of the world's rejects like the United States of America?

(iv.) One of the most important subjects exercising statesmen is the need for population and every medical man of twenty or more years' experience realizes that the Australian is not breeding as he did and knows very well that this is not due to loss of prolificness but to artificial means. The provident practise it and the improvident do not; some may read for provident intelligent and for improvident unintelligent. There are exceptions, of course, but the intelligent couple with an income

under £1,000 *per annum* and no prospects of increase is not going to have a family of ten or twelve children, whilst the unintelligent will continue to breed indefinite numbers, leaving the State to which he contributes, nothing but a potential pauper offspring to become a burden on the intelligent taxpayer. Thus the intelligent do not escape the expense of a large family by restricting their own output, but have to support the comparatively useless progeny of the proletarian.

CEREBRAL VASCULAR LESIONS: THEIR VARIETIES, SYMPTOMS AND SEQUELÆ.¹

By HERBERT W. MOXON, B.A. (Cantab.), M.R.C.S. (Eng.),
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Western Australia.

I HAVE to talk to you this evening upon the subject of cerebral vascular lesions, but first of all I desire to express my appreciation at the honour which has been done to me by Dr. J. T. Anderson and this Branch of the Association in the invitation to deliver a paper on the occasion of the Annual Meeting. Of this honour I am, I can assure you, fully conscious and for it I am duly grateful. As regards my choice of subject I must remind you that for reasons which are known to you all, the circumstances of our meeting require that I shall restrict myself to a subject of a neurological or quasi-neurological character and so I have chosen one which I trust may prove of the most general and practical interest.

The subject of cerebral vascular lesions is a very wide one and naturally you will not expect me to cover it in one comparatively short paper. Neither shall I make any such attempt, the two objects which I have kept closely in mind in planning my paper, being to interest you on the one hand and keep out of the textbooks as far as possible upon the other. You will, however, permit me as a starting point to refresh your memory with a short classification of the different varieties of cerebral vascular lesions which have to be kept in mind.

VARIETIES OF LESIONS.

In the first place, all these lesions fall into two natural groups, primary and secondary. Primary lesions are always due to disease of the vessel walls, secondary lesions are the result of pathological conditions arising elsewhere than in the vessels themselves. As we shall be concerned this evening entirely with those of a primary character, I will dispose of the latter as briefly as possible.

Secondary Lesions.

Trauma.

There are three principal causes of secondary cerebral vascular lesions and the first is trauma, such as fracture or other injury to the skull and

¹ Read at a meeting of the Western Australian Branch of the British Medical Association on March 21, 1926.

intracranial contents. As these lesions are purely surgical in scope and so outside my own province, I have nothing more to say regarding them.

Inflammatory Conditions.

The second cause of these secondary lesions is inflammatory conditions of the brain substance, typically found in such diseases as poliomyelitis and *encephalitis lethargica*. In these conditions, the lesion starts as a small round celled infiltration of the perivascular tissues and ultimately leads to thrombosis and hæmorrhage. When this stage is reached the state of things, so far as the cerebral vessels are concerned, is clinically indistinguishable from a primary vascular lesion.

Cerebral Tumour.

The third principal cause of secondary lesions is cerebral tumour, regarding which I have little more to say. For reasons associated with the peculiarities of the cerebral circulation generally all cerebral tumours and especially gliomata are very liable to produce sudden and profound vascular disturbances in their vicinity and the younger the patient, the more likely these disturbances are to occur. When they do occur, they result in a sudden and often alarming increase in the patient's symptoms, a condition of things often attributed to the tumour itself whereas the true explanation lies in a vascular disturbance in its neighbourhood. Often again these symptoms subside almost as suddenly as they arise and it is this liability to vascular disturbances in the vicinity that explains why so many patients with cerebral tumour improve so greatly under iodide of potassium, even when no syphilitic factor is present. So much for a brief survey in passing of secondary cerebral vascular lesions.

Primary Lesions.

We are now free to turn our attention to those primary cerebral vascular lesions which are the subject we are to consider tonight. These lesions are, as I have already mentioned, always due to disease, an arteritis of some kind, arising in the vessel walls or in the case of an embolus elsewhere in the cardiovascular system.

Thrombosis and Hæmorrhage.

There are two preliminary points in regard to these conditions the importance of which can hardly be overestimated, because they lie at the very root of a sound understanding of all primary cerebral vascular lesions. The first is that it matters not at all what the causal factor in these lesions may be, syphilis, arteriosclerosis, acute infections and so on, they all have the same end result, namely the production of thrombosis and hæmorrhage. The second point is that cerebral hæmorrhage and cerebral thrombosis are not antagonistic and mutually incompatible conditions between which it is essential or at times even possible to make a differential diagnosis, but on the other hand are conditions which from a pathological and clinical point of view, though not always from that of the patient, are but different stages in one and the same

process. Both are due to the same causes which may produce either result indifferently and all one can say is that in a patient who has had several strokes and ultimately dies from one, the earlier seizures are usually thrombotic in nature and the terminal one hæmorrhagic. Lastly, I should like to remind you how any infective condition of the blood can at any age lead to thrombosis and hæmorrhage of the cerebral blood vessels. Typhoid, pneumonia, influenza, whooping cough can all do it quite easily and in every case the causal organism can be recovered from the clot. None of us are in the least surprised if a patient with typhoid develops a femoral thrombosis, and we ought not to be surprised if one with pneumonia develops a cerebral thrombosis. And now a word as to the mechanism by which hæmorrhage and thrombosis are brought about. The first step, of course, consists in the appearance of a thrombus blocking the lumen of a vessel and as a result two local changes occur: (i.) The vessel wall itself undergoes degenerative changes and so becomes weakened. (ii.) The brain substance, dependent for its blood supply upon the particular vessel involved, undergoes softening and necrosis. Next, the necrotic brain tissue becomes gradually absorbed, the weakened and thrombosed artery is left practically unsupported and in consequence usually undergoes an aneurysmal dilatation. When this stage is reached, it is but a step to its eventual rupture as a result of some sudden exertion or rise in blood pressure. I shall return to the subject of thrombosis and hæmorrhage later, but I now pass on to the other varieties of primary cerebral vascular lesions which have to be kept in mind.

Embolus.

Cerebral embolus, the next lesion to which I will direct your attention, is nearly always associated with mitral stenosis, but occasionally with some other variety of cardio-vascular disease. It gives rise to an immediate and severe hemiplegia which may be very extensive and one needs to exercise great caution in giving a prognosis in these cases as almost anything may happen. Some patients manifest a sudden and rapid clearing up of all symptoms owing to a rapid contraction of the clot and a recanalization of the artery. *Per contra*, the clot which is of course essentially a thrombosis, may spread and in these cases recovery does not occur. In others again the clot may shift with a sudden clearing up of one set of symptoms and the equally sudden appearance of another. Thus a hemiplegia of one day may be followed by a monoplegia the next. Lastly, there is always the causal condition itself to be taken into account and this may and often does actually dominate the clinical picture.

Cerebral Aneurysm.

The next and last type of primary cerebral vascular lesion to which I desire to draw your attention, is a very interesting condition of which there are several varieties, namely cerebral aneurysm. First we have that quite unimportant variety known as miliary aneurysm which consists of small seed like

dilatations on the finer cerebral arteries in the substance of the brain. They are usually multiple and are generally associated with a widespread cerebral arterial degeneration; they never rupture and they are of no practical importance apart from the causal condition.

Secondly, we have those larger irregular dilatations of the cerebral arteries which are of frequent occurrence in connexion with syphilis and atheroma. They are of the nature of false aneurysms, usually of the "dissecting" type and are a not infrequent cause of severe and fatal hæmorrhage.

But the real interest of cerebral aneurysms arises in connexion with those of the true aneurysm type. Of this type there are several causes and varieties, but I intend to direct your attention to only two, the congenital and the syphilitic.

The congenital variety is probably due to some developmental defect in the vessel walls. It is met with *post mortem* at all ages from infancy upwards and apart from an infective arteritis is the principal cause of cerebral hæmorrhage in children. In size these aneurysms vary from that of a pea to a cherry and they are often multiple. They are most frequently found in the vicinity of the circle of Willis. They may rupture and are an important cause of cerebral hæmorrhage. But the really interesting thing about them is their peculiar tendency to leak; when they do leak it is into the subarachnoid space with a resultant clinical picture very different from that usually associated with cerebral hæmorrhage. And what happens is this:

In the first place, fibrin and clot are deposited around the nerve roots of the medulla and upper portion of the spinal cord. The patient complains of pain and stiffness in the neck and shoulder and the condition is diagnosed as rheumatism. This we may call Stage I. Next, the continued slow leaking gradually increases the intracranial pressure and headache, vomiting, head retraction and Kernig's sign make their appearance. The symptom complex at this stage which we will call Stage II., exactly resembles that of meningitis and this revised diagnosis is usually made. Finally, convulsions may occur and death result from a great rise in intracranial pressure consequent upon a blockage of the foramen of Monro. But there is generally one very suspicious point about this diagnosis in that there is no rise in temperature. This, of course, gives the clue to the real nature of the condition which may be determined by a lumbar puncture. Lumbar puncture yields a blood-stained cerebro-spinal fluid, red for the first day, then brown and after the fifth day yellow, if no further hæmorrhage has taken place. Lastly, a differential count of the white cells of the blood which are present in large numbers in the cerebro-spinal fluid, reveals an exactly similar proportion of the different varieties to that found in the blood itself. This, of course, excludes meningitis absolutely and settles the diagnosis beyond all doubt.

The second variety of true aneurysm to which I have to refer, is that due to syphilis. This variety

occurs as a rule on the larger cerebral arteries, particularly the basilar and incidentally gives rise to the largest aneurysms found in connexion with the cerebral vessels. These aneurysms may do one of two things: They may rupture with all the signs of severe cerebral hæmorrhage or they may not rupture and then on account of their large size give rise to the symptoms of cerebral tumour pure and simple.

In conclusion, the last point which I have to mention in connexion with cerebral aneurysm is that a large proportion of all cases give rise to no symptoms at all and are unsuspected during life or only found *post mortem*.

Atheroma and Arteriosclerosis.

You will notice that I have said nothing so far about primary atheroma and arteriosclerosis. They are of course the preliminary condition, often lasting for years, of primary vascular lesions which have thrombosis and hæmorrhage as their end results. The reason I think is obvious; for the subject is one which for adequate handling would require a whole or several papers to itself and at that I must leave it except to enter a plea in passing for its earlier clinical recognition when much can be done for it. The early symptoms of this condition are entirely due to an ischæmia of the brain and a typical story is somewhat as follows: A business man hurries for his morning tram or train and on arrival at his office finds a difficulty in mentally concentrating upon the work which he finds awaiting him. At lunch time, thinking he must be a little off colour, he turns to the worst possible remedy in his case, namely alcohol, as a result of which he feels rather worse than he did before. Towards evening the symptoms clear away, only to be repeated at intervals later until with a more or less permanent rise in blood pressure they may eventually disappear. What happens in these cases is, that owing to the peculiar physiological features of the cerebral circulation the blood supply of the cerebral hemispheres cannot properly adjust itself to the vascular changes and a relative ischæmia results. Upon an early recognition of this condition, whether in the course of an ordinary clinical or life insurance examination, depends the ultimate fate of the patient and your successful handling of the case.

SYMPTOMS AND ANATOMICAL CONSIDERATIONS.

I have now reached a point beyond which I cannot proceed without a brief digression of an explanatory character and it arises in connexion with the second subheading to the title of my paper, namely, the symptoms to which cerebral vascular lesions may give rise. Let me say at once, however, that I trust you will absolve me in advance of any thought of coming here this evening to drag you through the dull and interminable lists of symptoms which may result from a cerebral vascular lesion. You will find such lists in the textbooks, from which they cannot be omitted and they are all of but little use unless you hold the master key to their interpretation. What I am going to do on the other hand, is to use this opportunity to talk to you in

an informal manner about symptom complexes in general in connexion with nervous system lesions, illustrating them where possible by reference to cerebral vascular lesions in particular; for in this way I hope that I may succeed in interesting you and incidentally show you the loose end of the thread of that Ariadne's web in which most of us get entangled at an early stage in our study of the central nervous system.

To begin with, symptom complexes in connexion with nervous system lesions are little more than a confused jumble of disconnected facts until interpreted in the light of the fascinating research story of the past few years. I only wish that time permitted me to retell that remarkable and even exciting story by which chaos has given place to a large measure of order in regard to the form and functions of the central nervous system. Those of you who are acquainted with it, will know exactly what I mean, but for the benefit of those who are not, I must summarize as briefly as I can its main conclusions. First of all, however, I must find a starting point and so I will ask you for a moment to throw your minds back with me to our student days to the chaos to which I have just referred and I will give just one simple example of this chaos from the motor and one from the sensory functions of the central nervous system.

As regards the motor functions of this system we were taught by the physiologist that all movements were brought about by means of nervous impulses originating in the cerebral cortex which reached the periphery by means of the great corticospinal or pyramidal tract; but it was also impressed upon us that under no circumstances could this motor mechanism function until its projection fibres became medullated. Then came the embryologist who told us that for the most part medullation of this tract did not take place until after birth and what was even more puzzling still, that it had not even acquired a great part of its axis cylinder processes at the eighth month of the foetal life. How then could a foetus show vigorous movements at the fifth month of intra-uterine life—a fact which no one could possibly deny?

Next, we were instructed that that portion of the cerebral cortex immediately behind the fissure of Rolando was "sensory" in function; but when, as was done on several occasions, this part of the cortex was electrically stimulated in a conscious patient, never a sign of any kind of sensation with which we were acquainted was evoked by this procedure.

Finally, to turn for a moment to the clinical aspect of the problem, whence came the involuntary movements in a totally hemiplegic limb and in a patient with *paralysis agitans* or those repellent athetoid movements in a case of cerebral diplegia? Here then are two or three examples drawn at random from problems in connexion with the central nervous system which were not recondite and occult, but obvious, simple and elementary problems, quite inexplicable on the conceptions then held in regard to the form and functions of the nervous system.

And then the light began to dawn and if it can be said to have proceeded from a single sun, that sun was Sir Charles Sherrington to whose name there stands in my very humble opinion the most masterly series of researches in the whole history of physiology. And what we owe to Sir Charles Sherrington in physiology, we also owe in generous measure to one of my own teachers in clinical medicine, Henry Head. Now the only result of their work with which we are concerned this evening, is that master key to which I referred a little time back in connexion with the central nervous system and it is that, leaving aside the sympathetic, we possess not one nervous system but three. Regarded as a whole, the central nervous system is a true trinity; that is to say, it is three in one and one in three or, in other words, whilst a trinity in structure it functions as a unitary system so long as all parts of it remain intact, but no longer.¹

In this connexion the following essential conclusions may be emphasized:

In the first place the present central nervous system consists of three separate systems woven into one as follows:

- (i.) The simple spinal reflex, representing the simplest anatomical and physiological basis for the manifestation of nervous energy which concerns the *vertebrata*.
- (ii.) The great basal ganglia and their connexions which are a complete primitive nervous system—motor, sensory and equilibratory—used by evolving man before he acquired his cerebral hemispheres.
- (iii.) The cerebral hemispheres which, so far as we know, are Nature's creative masterpiece in the physical world.

In the second place each of these systems is actually or potentially complete and perfect in itself for the particular stage of evolution for which it was designed or at which it came into existence.

Thirdly the essential thing to grasp in connexion with these three systems is not their points of similarity, but their points of contrast. Thus the sensory portion of the second system, that is to say the optic thalami, are concerned with those primitive sensory apperceptions without which it is inconceivable that organic life could ever have evolved on this earth to any high level, namely coarse non-discriminative sensations of pain and temperature and emotional tone-feelings of pleasure and pain. The sensory functions of the cerebral cortex on the other hand are essentially discriminative and the cortex as a whole is the organ of the will. It would perhaps be better to drop altogether the use of the word "sensory" in connexion with its functions.

In the fourth place as adult life proceeds, naturally acquired combinations of complex motor movements, once they have become perfectly integrated by the cortex through ceaseless repetition,

¹ At this stage Dr. Moxon gave a demonstration of diagrams which he had prepared so that they might be superimposed on one another. The diagrams depicted the three systems described in the paper.

are probably relegated to the *corpus striatum*. Thus the youth of sixteen certainly shaves through the medium of his cortex, the man of forty probably through his *corpus striatum*. This frees the cortex for work of a higher character and above all the acquisition of new accomplishments, the ultimate purpose of which is an expansion of consciousness. This in its turn leads to reciprocal and ever-increasing demands upon and therefore added development of the cortex itself, a kind of circle of an entirely non-vicious kind.

Fifthly, when we have grasped in regard to the cerebral hemispheres which constitute the third system, that its motor functions are voluntary and initiative and probably operated by an inhibitory type of mechanism, a perfect contrast between it and the motor functions of the second nervous system (the basal ganglia) is given in the following words of Sir James Purves Stewart:

All movements in a new-born infant are either reflex or automatic; they are pre-ordained and admit of no choice. Only gradually does the child learn to use his cerebral cortex, to call in antagonistic muscles, and, by an effort of the will, to inhibit reflex and automatic acts, to initiate voluntary ones, and thereby consciously to adapt himself to his environment.

It should now be quite clear that many lesions of the nervous system give rise to a symptom-complex, partly due to an interference with the functions of the system actually involved in the lesion and partly to "release" or "over-action" phenomena on the part of another system which remains intact. The classical example of this is found in cerebral diplegia. In this condition, owing to its more or less complete escape from cortical control, the *corpus striatum* discharges those involuntary impulses which give rise to the reflex and automatic movements called athetosis. Similarly, the rigidity and exaggerated knee jerks found in an old hemiplegic patient are due to the loss of cortical control over the mechanism of the *corpus striatum* and simple spinal reflex. One could, of course, multiply such examples almost indefinitely, but I must return to the more strictly clinical aspect of my subject and illustrate the foregoing by pointing out the main symptoms associated with a cerebral vascular lesion at a few chosen points, always bearing in mind that symptoms due to an interference with one system are likely to be associated with release phenomena due to over-action of another.

A lesion in the motor area of the cortex, unless it be due to an extensive injury, rarely gives rise to a hemiplegia owing to the wide area of the cortex devoted to this function. Thus a monoplegia is what we will expect from a vascular lesion in this neighbourhood. If such lesion lies behind the fissure of Rolando one would look for disturbances in light touch and such aspects of sensory discrimination as size, weight, shape and texture, all of which tend to be disturbed together in lesions involving this part of the cortex.

Coming down to the internal capsule where everything is so crowded together, we get our classical hemiplegia of the voluntary type when the lesion

involves the neighbourhood of the *genu*. If the posterior limb is involved, there will be signs of hemianæsthesia, usually associated with a partial hemiplegia, a monoplegia or a hemianopia, because a lesion in this situation is rarely so localized as to involve the sensory fibres of the capsule only.

A unilateral vascular lesion at the level of the *crus* gives us Weber's syndrome, that is a crossed paralysis consisting of hemiplegia of the opposite and a third nerve palsy of the same side. A unilateral lesion in the *pons* at the level of the seventh cranial nerve gives us another crossed paralysis, namely a hemiplegia of the opposite and a facial palsy of the ipsilateral side; whilst a similar lesion below this level in the *pons* or *medulla* gives a hemiplegia of the arm and leg only of the contralateral side.

The last type of hemiplegia to which I will draw your attention, is the so-called carotid hemiplegia, because it gives rise to a symptom-complex quite unique in clinical medicine. It consists of a hemiplegia of the opposite side and blindness upon the same side as that of the lesion. The former is due to the cutting off of the blood supply through the middle cerebral artery and may rapidly clear up owing to the establishment of a collateral circulation; the latter is due to obstruction of the blood supply through the ophthalmic artery. As the thrombus always spreads into this vessel, the blindness does not disappear.

Let me now direct your attention to the three principal clinical types of patient who are the subject of cerebral vascular lesions. First, we have the patient who is the subject of a frank hemiplegia. Regarding this type I need say nothing more, as it is so familiar to you all. Next, we have that type of patient who has repeated, slight, transitory attacks of thrombosis, producing for example a temporary dysarthria and culminating in a definite clinical picture of pseudo-bulbar palsy. You have all seen these patients and been struck over and over again with what is indeed a constant and permanent feature of them—the sudden and repeated passage from a bright cheerfulness to an intense lachrymosity. You may notice this cycle repeated three or four times in the course of half an hour's consultation and next time you witness it you will know the reason is that the optic thalami have been partially released from cortical control by the thrombotic lesions and the primitive uncontrolled emotional tone feelings are finding their inevitable expression. In a word, the patient has become a small child again in this respect, except that he has gained and then lost as a result of his malady what the small child has yet to acquire in the first instance. Lastly, we have the third type of patient who presents much the same clinical picture as the second, but without such markedly lachrymose tendencies. He gives evidence of considerable rigidity, slight tremor and has a short shuffling gait almost suggestive of *paralysis agitans*. This type is produced by scattered areas of softening and necrosis due to thrombosis in the neighbourhood of the basal ganglia and it is the condition which the French describe as *status lacunatus*.

I need refer but briefly to lesions directly involving the *corpus striatum* and optic thalami. The two dominant characteristics of *corpus striatum* lesions are rigidity and involuntary movements. If the lesion involves the large ganglion cells of the *globus pallidus*, we have muscular rigidity with tremor and disturbances of automatic movements such as the winking reflex and associated movements such as the swing of the arm in walking. This clinical picture is typically seen in *paralysis agitans*. If, on the other hand, the lesion principally involves the small ganglion cells of the neo-striate system which are contained in the *putamen* and caudate nucleus, we have gross interference with the limited coordinating power usually exercised by these higher evolved cells over the lower. The result is typically seen in the choreiform and athetoid movements associated with Huntington's chorea.

The thalamic syndrome is well known, but a little complicated and time will not permit our study of it in any detail. Hemianæsthesia to touch involving half of the body, intractable paroxysmal pain amenable to no known drugs, hemihyperæsthesia to temperature and pain, emotional tone feeling release phenomena, hemiataxy on voluntary movement and involuntary movements due to interference with the thalamostriate connexions—any of these separately or in combination may constitute the thalamic syndrome in any particular case.

Lastly and for the sake of completeness, if any of you are disposed to think that I have strained the facts in giving to the simple spinal reflex independent status as the first nervous system, I will only ask you to study the phenomena associated with the mass reflex and I venture to think they will soon convert you to the point of view taken up in my paper tonight. I do not suggest, because I do not believe, that evolving man ever had a nervous system consisting of an indefinite number of isolated, automatic, but perfectly developed reflexes as we understand that term today, any more than I believe that he ever possessed a nervous system which anatomically and physiologically was anything more than the prototype of the basal ganglionic system as it exists today. To me such a proposition would be quite unacceptable, because Nature never supersedes without a profound modification of that which is superseded, in order that it shall fit in with a fresh scheme of things. Rather my contention is that the spinal reflex and basal ganglionic systems are modified and reconstructed remains of preexisting nervous systems which once possessed independent functioning power; that in their modern and reconstructed forms they are still capable of functioning independently today under pathological conditions and that the phenomena associated with the mass reflex prove that the spinal reflex is the reconstructed remains of a nervous system which once enjoyed freedom and independence before the basal ganglia came into existence. In face of Gaskell's discovery that the central canal of the spinal cord is the remains of an old alimentary canal, it appears to me that such a view presents no difficulties as regards either understanding or acceptance. Nature may abolish whole types, as presumably occurred

with some of those which existed in antediluvian times. I doubt if she ever really abolishes structures and organs in a type which has proved its fitness in the evolutionary struggle, but rather modifies and reconstructs them to subserve new functions as the type itself progresses. Who can say what at present undreamed of functions the vermiform appendix may not develop in the course of the next few million years?

SEQUELÆ.

The last aspect of my subject this evening has to do with the sequelæ of cerebral vascular lesions and I propose to consider these as briefly as possible from the standpoint of prognosis and treatment.

In the first place, practically every case of cerebral hæmorrhage is preceded by thrombosis and of all patients with cerebral hæmorrhage 90% die within the first twenty-four hours with progressive symptoms due to hæmorrhage into the cerebral ventricles and consequent enormous rise in intracranial pressure. In children such hæmorrhage is usually due to rupture of a congenital aneurysm or else to an infective arteritis. In the elderly it is due to arteriosclerosis and as a rule is secondary to thrombosis. In early adult life it is usually due to syphilis and in this case it is always secondary to a pre-existing thrombosis. In my experience, when you take into consideration the widespread prevalence of cerebral-vascular disease, I do not think that cerebral hæmorrhage of this kind is very common; but when it does occur, it is nearly always fatal within a few hours.

As regards thrombosis and the remaining 10% of patients with hæmorrhage the immediate outlook is, of course, far better; but a differential diagnosis may be extremely difficult and indeed impossible by clinical observation alone. If the onset occurs during sleep and is preceded by prodromal symptoms such as headache and vertigo, the lesion is probably a thrombosis. If it occurs after exertion, a previously thrombosed vessel has probably ruptured. If you must make a differential diagnosis, which is not a matter of great importance, you must do a lumbar puncture; for in practically every case of cerebral hæmorrhage the cerebro-spinal fluid is blood stained within the first twenty-four hours. As regards prognosis in the case of cerebral embolus I have already pointed out the pitfalls here when referring to this condition. May I now give another warning in regard to the prognosis in cases of syphilitic thrombosis, especially those occurring in early adult life? This condition is usually associated with an acute encephalitis of the neighbouring brain substance and there is much lymphatic extravasation as well as local œdema due to an evasulation caused by the thrombosis. The symptoms, therefore, are not all due to the thrombosis and are amenable to specific treatment. In consequence one requires to exercise due caution in regard to giving a too unfavourable prognosis. Lastly, the key to a correct ultimate prognosis in a case of frank hemiplegia is given by the state of things as regards a return of the finer movements. All of us must have witnessed

the rejoicing which takes place when a patient with a thrombotic hemiplegia shows a definite and rapid return of voluntary movement in the paralysed limb and in this rejoicing we may and ought to share. But when it comes to regarding it as an earnest of what is to come, it is quite another matter. In a case of this kind in which a rapid return of voluntary power as regards the coarser movements occurs without a corresponding improvement in the finer discriminative movements, the ultimate outlook from a functional point of view is not good. Therefore always ask such a recovering hemiplegic patient to remove a match from its box and strike it, using the paretic limb for this purpose, for here you have a far more reliable guide as to the ultimate degree of recovery to be expected than is the strength of his grip or his ability to walk unaided.

I suppose I may safely say that no one present here tonight will question the statement that at best the sequelæ of cerebral vascular lesions are generally speaking bad. That they are often made worse by wrong or indifferent treatment is nothing short of deplorable. And here let me begin at the beginning. In the 90% of hæmorrhage cases which prove fatal within a few hours all medical treatment is futile and useless and our only future hope here lies in surgery, a hope which we owe largely to Harvey Cushing. A few patients are temporarily benefited by lumbar puncture which should be performed for diagnostic purposes and this procedure may and not infrequently does lead to a return of consciousness, a point which may be of some importance. Next, the treatment in all cases of apoplexy should be stimulant. To adopt depletive measures and measures directed towards lowering the blood pressure and diminishing the force of the cardiac action is simply to ask for further trouble, as a little reflection on the peculiarities of the cerebral circulation will quickly make clear. Finally, we have to consider the position and sequelæ in a patient who has sustained and survived an attack of cerebral hæmorrhage or thrombosis. Here the first thing to do is to look ahead and the first thing to look at after the patient himself is the joints of the paralysed limb. Over and over again one sees old hemiplegic patients with a fair return of voluntary power in whom, however, any attempt to move the affected limbs provokes such extreme pain in the joints that such movements are at once stopped. This is due to the "rest" adhesions which so quickly form in a paretic limb. The pain which they cause, is really very severe and it is all preventable by regular passive movement of the joints from the very outset. With returning power, massage and passive movement should be daily applied to the affected limbs as a whole. The one thing to avoid is electricity in any shape or form. The constant current is useless, the faradic is severely harmful; for it definitely increases spasticity which is the very last thing to be desired.

The next sequela to be avoided as far as possible is deformities and this you can only hope to do by incessant watchfulness and ingenuity in availing yourself of the various mechanical appliances and

reeducative methods which are at your disposal. Details regarding these you will find in the textbooks. An enormous amount can be accomplished with time and trouble towards the prevention of these deformity disasters, but you will be more than repaid if your patient eventually shows a good degree of voluntary recovery. Those in charge of him should be instructed as to the abnormal and deformity-producing positions which the paralysed limbs must not be allowed to assume and they must if necessary be furnished with suitable means to this end.

Last and I ought to have put it first one has especially in these cases to remember to treat the patient first and the disease afterwards. Practically all these patients suffer from severe depression, especially at the onset of their condition and it is our duty to combat that depression by instilling into them encouragement and hope by every means in our power. Point out from the very beginning that the initial paresis is practically always more extensive than the residual. As soon as there are any signs of returning voluntary power, see that it is utilized in the right way to encourage the patient on the one hand and aid a further recovery upon the other. Keep your patient active when activity is possible. Get him up on his feet as soon as he can stand. Utilize and invent exercises and measures which will keep him occupied and reeducate him to a new activity. Refrain from all attempts to reduce a blood pressure below say two hundred millimetres of mercury, thinking to avoid a further stroke, because attempts of this kind are the surest way to precipitate the very thing you are trying to avoid. Do all these things or refrain from doing them, as the case may be and you will do much, very much, towards the prevention of some of the worst sequelæ of cerebral vascular lesions, because you will not add the avoidable sequelæ of indifferent treatment to the unavoidable ones of the causal condition.

And now it only remains for me to offer you my sincere and grateful thanks for the kind and patient hearing which you have given to me this evening.

THE SEQUELÆ OF LETHARGIC ENCEPHALITIS.¹

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THE output of literature upon the subject of epidemic encephalitis and its after-effects has been considerable. In view of the fact, however, that sequelæ are continually being seen and are at times difficult to recognize, it may be of some interest to review the records of patients who have been under observation for varying lengths of time. Many of

¹ Read at a meeting of the Section of Neurology and Psychiatry of the Victorian Branch of the British Medical Association on November 25, 1925.

the patients under consideration have been presenting themselves during the past two and a half years at the Neurology and Psychiatry Clinic at the Melbourne Hospital. Some are under observation at the Austin Hospital.

In nearly 80% the condition had not been diagnosed in the primary stage of the disease. The epidemics of a few years ago were more widespread and the sporadic infections more frequent than was apparent at the time. Close inquiry into the clinical manifestations of the primary condition makes it difficult to see how the clinician could have distinguished between influenza in one of its manifold forms and an attack of apparently mild epidemic encephalitis.

In these "influenzal" forms lethargy does not seem to have been present at the time, although many of the patients complained that they had been drowsy after the more acute symptoms had disappeared. Diplopia had not been present according to the patients' statements at the time of the primary disease. Many said that they had been sleepless and inclined to be mildly delirious at night. Pains in the limbs had been complained of by some of them. In two patients the primary symptoms were so mild that a medical practitioner had not been consulted.

When they first presented themselves, many of the patients complained of vague subjective nervous symptoms which on their face value could have been mistaken for those of neuroses. Objective physical examination often reveals very little neurological information.

The mistake has often been made in thinking that the patient's demeanour and expression is indicative of their state of mind. Thus these patients have been thought to have been "depressed" or "stupid," leading to an erroneous diagnosis. As will be seen presently, a careful study of the patient's facies will often give a clue to the diagnosis.

In this paper I propose to review quite briefly the cases themselves. Then I shall discuss the symptomatology and what is known from observers about the mechanism involved in the production of such symptoms. The first case may be described more fully than the ensuing ones, as it embodies in its most typical state the syndrome that is occurring to a greater or less degree in all the patients. Moreover, this patient takes an intelligent interest in his own symptoms and some information as to the mental state occurring in these patients can be gauged from his own introspection. This case was not diagnosed in the early stages of the disease.

CASE I.—This patient previous to his illness was an expert tailor employed by a well known firm. In 1921 he contracted "influenza" in which he was drowsy by day and restless by night, sometimes delirious. Very shortly after an apparent recovery he began to find that he was unable to work, as he could not carry out properly the movements that his work entailed. I saw him first in June of 1923, when he had the Parkinsonian syndrome as he has at the present time. Physically his condition had been quite stationary for the past two years.

In regard to his attitude and gait he stands with slightly stooped shoulders and holds his head somewhat stiffly. He

walks perfectly well, but does not swing his arms when walking.

His facial expression is not fixed, but somewhat immobile. His eyes are inclined to be staring and there is some abnormal moisture about the eyes. He has the characteristic smile, slow to appear and slow to disappear; the skin of the face presents the slightly greasy appearance, described by Wimmer.⁽²⁾

There is a definite limitation of the movement of his eyes in all directions, especially in an upward direction. This does not mean that he is unable to move his eyes in either direction, but it is a considerable effort for him to do so. Thus his eyes stare straight in front of him and the inclination is to turn his head when looking round instead of simply moving his eyes. There are no pupillary changes.

His voice is monotonous in tone; at times in speaking it fades away or he may pause for a few moments as though he were stumbling over a word.

There is a slight, generalized muscular rigidity of the cog-wheel type affecting the limbs. This seems to be pathognomonic of the post encephalitic syndrome and cannot be simulated by hysteria. Voluntary movement is performed slowly, but without any loss of power, although the power easily tires unless the patient's attention is kept upon what he is doing. On urging the patient, movements can be performed quickly, but it obviously takes some effort on his part.

The reflexes are normal.

This patient manifests the Parkinsonian syndrome, better expressed as the bradikinetik syndrome, in its most typical state. It is to be noted that although there is a disability or difficulty in performing movements, there is no true paralysis. The patient is able to carry out any movement that he wishes to perform, but there is a general disinclination to do so, as it is not easy for him.

Discussion.

Subjective Symptoms and Mental State.

At first appearance this patient seems dull and stupid. After a short conversation with him, this impression is removed. He answers questions slowly, as though he were thinking of what he were going to say. His answers are pertinent and he gives a good account of his own symptoms and subjective feelings. He complains that he feels drowsy during the day time and is unable to keep his mind fixed for any length of time upon any work that he is trying to do. His inability to do sustained work is due to the mental and physical exhaustion that ensues when he attempts to carry out the movements appropriate to his trade (tailoring).

He is able to do the work accurately and well, as long as he does it for series of brief periods. For instance, he has recently made himself a suit of clothes, but it took him some months to make them.

He notices this disability of movement in everyday acts, such as dressing. There have been times when in the act of taking his food to his mouth his hand will stop half-way between his plate and his mouth and will remain in that position for a few minutes, until his attention is drawn to the attitude. This, he says, happens when his mind wanders to other subjects and he "forgets what he is doing." In other words his attention process has to be rivetted to whatever muscle action he is carrying out.

He does not complain of stiffness of his muscles, but of the inability to swing his arms in walking, not because there is any resistance to be overcome, but because there is an inability to do so automatic-

ally. In fact he is quite clear about all his slowness in movement being due to the fact that "something is not there to make him do things without effort." Sometimes he will sit for hours at a time in one position, staring in front of him. This is like a condition of catatonic stupor, and a catatonic type of this condition has been described.⁽¹⁾ However, there is not any real relation to true catatonia. He says that when he is like this his thoughts are perfectly normal and he feels restless; after a time he begins to feel drowsy. There is obviously no schizophrenia nor is there any sign of a delusional state. These symptoms vary considerably from day to day. On some occasions he feels well and interested in what he is doing and on these occasions he is able to do things much more easily than at other times.

It is this variation of symptoms that may make the observer think that there is a possibility of a functional element in his behaviour. I have seen this patient in moments of excitement carry out movements almost as rapidly as a normal person. His own people were perfectly frank in saying that they thought that half his complaint was due to the fact that he did not want to do any work.

On closer examination of this patient it is found that all movements of the limbs can at any time be carried out quickly and strongly, as long as the patient's attention is fixed upon what he is doing. Immediately his attention begins to wander, the movements themselves begin to become weaker and less sustained.

Before the onset of the primary disease, this patient was a moderately keen reader. He now says that he is unable to read, not because he cannot take in what he does read, but because the effort of following the print with his eyes is too great to be sustained for any length of time.

It can be judged from the foregoing symptoms that the patient's easiest position is the sitting position. This, he says, is so and his people say that he will sit for hours in a corner "moping." In reality he is not miserable or depressed at these times, but feels calm and restless. It is when he is trying to do things and is unable to do what he wants to do comfortably that his misery and depression are most poignant.

This patient's emotional feelings are to some extent in abeyance. He smiles fairly easily, but he says himself that his smile does not necessarily mean amusement. Some of these patients have the same facility for weeping without feeling the emotional tendency to weep.

He has been taking hyoscine hydrobromide 0.6 milligramme (grain one-hundredth) by mouth nearly continually since observation. This lessened the rigidity that was present, and altogether he is more comfortable. There have been no apparent ill-effects from the drug and if by chance he does leave off the drug even for a short time, he feels the effect.

CASE II.—The second patient, a male aged twenty-six years, presents the picture of a gross Parkinsonian syndrome, without the typical rhythmical tremor.

When first seen he was continually dribbling saliva from his half-open mouth. This was checked by the administration of hyoscine by mouth. He has definite "cog-wheel" rigidity. He complains of being unable to read as the print is always blurred. His pupils react only sluggishly to light and scarcely at all to accommodation. This pupillary change has been noted by Wimmer⁽²⁾ and Duncan⁽³⁾ on occasions. His mental attitude is one of general inertia, although his intelligence is good. The symptoms came on a month after an attack of influenza. A Wassermann test did not yield a reaction.

CASE III.—The patient, a male, *etatis* thirty-two years, is almost a replica of Case II. for the absence of any pupillary changes. His mental attitude is one of euphoria. He is at the Austin Hospital, where his life flows along easy channels. It is the sense of inferiority to his fellow creatures that often brings a train of psychoneurotic symptoms into prominence. This is seen in the patients with milder conditions, who have been treated as out-patients, more especially if they have been treated along functional lines.

The next four cases are of the nebulous type in patients who complain mainly of subjective feelings of insufficiency. They are unable to work; the inferiority feelings are often evident. What is referred to as the bradykinetic syndrome, is present to a mild degree. The facies may mislead the observer to think that the patient is depressed or dull and that he is suffering from neurasthenia, psychasthenia or a high grade amentia.

CASE IV.—The patient, a female, aged twenty-three years, contracted influenza six years ago and has not been well since. It was only after some difficulty that the connexion between the influenzal attack and the present illness could be established. This patient was sent from the country with a provisional diagnosis of "neurasthenia" or "nervous exhaustion." Her complaint was that whenever she tried to do any housework she became quite exhausted. She had taken many tonics, which did not seem to improve her condition; she was feeling disheartened and depressed. Examination showed that there were some immobility of her features, together with an inability to wrinkle her forehead. There was a suspicion of cog-wheel rigidity in all her limbs. All her movements were performed more slowly than normal and she easily tired.

CASE V.—The patient, a male, aged nineteen years, has not been able to keep in employment for more than a month at a time, as he has been dismissed for incompetency each time he has secured a position. After some difficulty it was discovered that he had had "an attack of influenza" just after leaving school at the age of fourteen. He had been a good worker at school and had passed out of the highest grade. The boy has been regarded as stupid and incompetent and had been considered to be a high grade ament.

Careful observation showed that the eyes are slightly staring and moister than normal. There is a slightly greasy appearance of the skin, a slow smile, with some difficulty in wrinkling the forehead. There is slightly generalized cog-wheel rigidity.

CASE VI.—The patient, a female, aged thirty-two years, complains that she has not felt well since an attack of mild influenza in 1921. She feels that she is not like other people and that her housework is a great trouble to her. Lately she had developed symptoms of nervousness, feels that her memory has gone and has a general feeling of inferiority.

Her physical signs are unilateral. The left side of her face is less mobile than the right side, especially in regard to movements of expression. She complains of dropping objects out of her left hand. There is a definite slowness in executing movements with that hand. There is also some cog-wheel rigidity of upper and lower limbs on the left side only.

CASE VII.—The patient, a male, aged thirty-four years, has been complaining for some time of pains in both feet;

the condition was regarded as probably a functional one. He had been in the Australian Imperial Force and had contracted mild trench feet in France, but the feet had felt well after a period out of the line.

It was noticed that this patient's face was the least immobile and that there was definite cog-wheel rigidity of the left upper and lower limbs. The history was that in the ship coming to Australia in 1919 he suddenly found that he was unable to sleep at nights. The medical officer put him into the ship's hospital and he seemed to improve. A day or two later he landed in Melbourne and went home. He began to be tired in the day time and sleepless at night. He went to a hospital where he slept for thirty-six hours and after that felt well and was allowed to go home. There is little doubt that this illness was a form of *encephalitis lethargica*. Whether the pain complained of in the feet was a remnant of trench feet or whether his general feeling of inferiority had engendered a "functional" pain in a part of his body that he knew had received injury, is difficult to say.

CASE VIII.—The patient, a male, aged forty-five years, is the only adult in this series showing definite psychotic symptoms. He had influenza in 1921. Previously he had been a keen and successful business man, but after the illness he was not able to work. At first he was depressed and his memory seemed to be failing. He could not concentrate his mind on any subject and he lapsed gradually into a condition in which he could only answer simple questions with difficulty, as he did not seem to grasp what was being said to him. His memory both for past and recent events failed. He cannot even remember the name of the firm for whom he worked.

Physical examination shows that there is definite general cog-wheel rigidity. There is a fixed immobile expression and his emotional reactions are in abeyance. All his movements are very slowly performed. Clinically he presents a picture similar to that of an advanced melancholia.

CASE IX.—The symptoms of this patient, a male, *etatis* thirty-eight years, also date from an influenzal attack in 1921. He was a railway worker and could not return to his work after his illness. He complains of tiredness during the day time, but is always restless at night. This symptom has been present ever since his illness and has only lately begun to disappear. He has been irritable, careless in his habits. He complains that his eyesight is poor. Mentally he is fatuous and somewhat childish, although his powers of perception and ideation are fairly good. Examination shows that his pupils are unequal and react poorly both to light and accommodation. There is a little secondary atrophy of the optic discs. Possibly his is a case in which there was a slight degree of papilloedema in the early stages. His features are immobile and there is a tendency to lurch towards the left when he walks. The reflexes are normal and there is no rigidity. The mental condition of this patient has greatly improved; he is now able to do some light work.

CASE X.—In regard to this patient, a female, aged twelve years, after some difficulty a history of influenza in 1920 was elicited. Until the age of seven the child was perfectly healthy and normal in every way. At this time she suddenly became wayward and difficult to manage and completely changed in temperament, although no physical signs were noticed at first. As time went on she became more difficult to discipline and would make no attempt to learn to read or write. It was then noticed that at times she would fall towards the left side and as she did this when she was thwarted in any way, this symptom was thought to be part of her general naughtiness. Later on a slight tremor of the left hand developed. This tremor was present only when she was using her hand or arm. At the same time some slight though definite cog-wheel rigidity was present in that arm. An inability to wrinkle her forehead was next observed and the tremor and rigidity became more evident. At present her condition has advanced considerably and she has now very definite rigidity in all four limbs, with the typical facial immobility. Her mental condition has remained unchanged, although as she is unable to do so much as she could, there is not the same difficulty in managing her.

CASE XI.—The patient, a female, aged twenty-eight years, had a definite attack of *encephalitis lethargica* in 1922. Physically she presents the appearance of a patient with very mild Parkinsonian features, with some bradykinesia and slight cog-wheel rigidity. Previous to being seen at the hospital clinic, she had been having many hysterical seizures, with phases of hysterical mutism between the fits. It was found out that she was living in somewhat difficult and uncongenial surroundings. When her environment was improved, her functional manifestations disappeared and she has been fairly well since.

CASE XII.—The patient, a male, aged thirty-six years, had a definite attack of *encephalitis lethargica* two years ago. This patient at present is extremely rigid, so much so that he is unable to get out of bed. His facial muscles are fixed in an expression resembling that of Wilson's lenticular degeneration, the spastic grin. His speech is affected and he can only talk with great effort. There is a great deal of muscle wasting throughout the trunk and limbs and there is considerable flexor contraction of the left wrist. At times he has urinary incontinence. This patient is bed-ridden and his mental state is difficult to gauge. His intellectual faculties appear to be good.

General Symptomatology.

In all these cases both physical and mental signs and symptoms are exhibited. In some the former predominate over the latter and *vice versa*. There appears to be little correlation between the severity of the two groups of symptoms.

Wimmer⁽²⁾ classifies the types of the disease as: (a) Parkinsonian, (b) intermediate type, (c) the hyperkinetic type. In the present series of cases there are none of the latter group, that is the type with choreo-athetoid movements. The diagnoses of all these cases rests upon: (i.) the clinical history and (ii.) the presence of bradykinesia and (iii.) the increase of muscle tone. The facies, monotony of voice and lessening of the so-called automatic and involuntary movements are all part of the slowness of muscle movement or bradykinesia. In some of the patients these physical signs are so slight and the subjective symptoms so great that the diagnosis is liable to be confused. Duncan⁽³⁾ in his analysis of a large number of cases states that residual cranial nerve palsies (especially the seventh and the third), monoplegias and hemiplegias must also be included in the physical findings of the sequelae of lethargic encephalitis.

The gross Parkinsonian types are almost akin to *paralysis agitans*. There are points of difference in the age incidence, clinical history and the fact that the typical tremor described by Parkinson is rarely present (Cruchet⁽⁴⁾). The increase of muscle tone is of a typical cog-wheel character, common to both *paralysis agitans* and the condition under discussion. Walshe⁽⁵⁾ thinks that the bradykinesia is directly dependent upon the increased muscle tone. Other observers do not agree with this statement. There are certainly clinical cases in which the syndrome is seen with very little hypertonus. Hyoscine will undoubtedly relieve the hypertonus, but this does not allow movements to be carried out more quickly. Of the mental symptoms Duncan⁽³⁾ states that defective memory and defective concentration of attention, drowsiness, alteration in temperament, hysteria and psycho-neurosis are the minor symptoms; mental deficiency, manic-depressive insanity

and dementia are the more serious sequelæ on the mental side.

Patho-Physiological Mechanism.

McAlpine,⁽⁶⁾ Wimmer⁽²⁾ and others have described the lesions in the *substantia nigra* and *corpus striatum*. McAlpine considers that probably the weight of the process falls upon the *substantia nigra* in epidemic encephalitis, whereas in *paralysis agitans* the *corpus striatum* bears the brunt of the disease. The *substantia nigra* is connected closely with the *corpus striatum*, but its connexions are not fully known.

It has been long known that motor muscle activity can take place without intervention of the pyramidal tracts. Sherrington⁽⁷⁾ first demonstrated that muscle tone was primarily for maintenance of posture of the limbs. The works of Magnus and de Kleijn,⁽⁸⁾ Riddock,⁽⁹⁾ Walsh⁽⁵⁾ and many others have tended to show that each physiological neural level that governs a posture maintaining reflex, is for the needs of the organism at that level and that a series of complex movements can take place at each of these levels, for example the spinal defensive flexor reflex. From a phylogenetic standpoint the *corpus striatum* appears to possess a higher integrating function than the nuclei in the spinal and brain stem. Muskens⁽¹¹⁾ has demonstrated a close connexion between the posterior longitudinal bundle and the *tractus interstitio-spinalis* and the *corpus striatum*. It would appear then that the *corpus striatum* was the integrator for certain movements of the eye, face and general somatic musculature. These movements would appear to be originally of an offensive or defensive character. Clinical evidence in man of this is borne out by the fact that in upper motor neurone lesions of the facial muscles movements in response to emotions are present, whereas the same movements cannot be voluntarily performed. Thus it seemed feasible to suppose that disease of the *corpus striatum* or its connexions was responsible for the syndrome that we are discussing, that is, elimination of non-voluntary movements.

Kinnear Wilson⁽¹³⁾ refutes this hypothesis firstly of all that there is no true paralysis. All the apparently non-voluntary movements can be carried out, although with an effort of volition. His second reason is that the *corpus striatum* in man is a relatively simple structure and that it is an absurdity to give to such a structure a function of such complexity. The main functions of the *corpus striatum* are to inhibit the tonus-maintaining centres in the mid-brain and to inhibit an inherent neuro-muscular rhythmical tremor low down in the physiological hierarchy. Wilson thinks that fibres go from the frontal lobe *viâ* the thalamus to the facio-respiratory muscle for facial expressional movements. He points out also that fundamentally all movements which involve consciousness to any extent whatsoever, must arise from the cerebral areas. Thus the Betz cell in the motor cortex is a relay between the higher cerebral areas and the mid-brain centres. The movements that have hitherto been designated as "involuntary," are not really so, as they are under

volitional influence. Notwithstanding Wilson's arguments it seems feasible to postulate that secondary impulses from the higher centres may be transferred to the thalamus and that the motor path from the latter may be along thalamo-striatal mesencephalic connexions, thus giving rise to "non-voluntary" movements. We knew too little of the function of the *corpus striatum* to do more than assume its functions in man.

With regard to the increased muscle tonus, Walshe⁽⁵⁾ has demonstrated by means of paralysing the proprioceptive nerve endings by means of "Novocaine" injections, that the existing hypertonus disappears. This shows that the increased muscle tone in *paralysis agitans* is of reflex origin, depending upon the proprioceptive nerve endings in the muscle substances for its afferent limb.

Hunter⁽¹⁴⁾ has brought forward the theory of dual mechanism for postural tone, comprising extensor contractile tonus under the influence of Deiter's nucleus and plastic tonus with its mid-brain centres in the *nucleus reticularis* of the *pons varolii*. Plastic tonus is considered by Hunter to be that met with in *paralysis agitans* and allied conditions. The *corpus striatum* has an inhibitory influence upon the mid-brain centres for plastic tone and its release gives rise to the overloading of the musculature with plastic tone. This is supplied by means of sympathetic fibres coming from the *nucleus reticularis*. Hunter considers that there is no contractile element in this plastic tonus.

The question is whether or not this slowness of movement is a symptom due to the release of striatal function alone. It would appear that the increased muscle tone is due to a release of striatal function, but the bradykinesia is not entirely dependent upon increased tone of the muscles. Wilson points out that the syndrome in question is due to an unwillingness on the part of the patient to participate in these movements, more than an inability to do so.

Wilson criticizes Hunter's hypothesis. He points out that there is a definite flexor attitude of the upper limbs and trunk. In other words, there must be some contractile element in this so-called plastic tone for the limb to take up the flexor attitude.

Walshe⁽⁵⁾ in criticizing Hunter's hypothesis considers that Hunter has gone too far in trying to postulate the dual components of postural tone and that experimental evidence does not bear out his hypothesis. The one patient with rigidity following encephalitis on whom I have seen operation performed, manifested no improvement. In reading the literature I am struck by the very few definite facts that are known about the functions of the *corpus striatum*. There appears, however, to be little doubt that the *corpus striatum* inhibits certain tonus-maintaining centres in the mid-brain. Hunter thinks that for voluntary muscle activity to occur postural tonus must first be inhibited by cortical control *viâ* the fronto-ponto-cerebellar tracts. In the case of the Parkinsonian syndrome this occurs, but as the inhibiting action of the *corpus striatum* is deranged, the excess of plastic tone remains.

Wilson, however, shows that the laws of reciprocal innervation are obeyed in these cases. The reciprocal innervation of the protagonist and antagonist muscles can be easily demonstrated in the patient. If the patient is asked to attempt to flex his elbow against a given force, the biceps will be tense and bulging, whereas it follows from this that the tonus possessed by the antagonistic group is inhibited in involuntary movement and is, therefore, not wholly "plastic" in containing no "contractile" element.

The cortical motor area is in reality merely a relay station between the higher volitional centres, presumably in the frontal cortex and the lower centres in the pons, medulla and cord. There is undoubtedly a definite conscious factor in the unwillingness to initiate and maintain movements that entail a considerable effort on the part of the patient. The *corpus striatum* has no known direct connexion with the cerebral cortex and so cannot be under the control of consciousness. It would seem that the lesion must be on the afferent side of the Betz cell area.

Psychology.

We have seen that in these patients there is an inhibition of volition to perform movements that in the normal person are carried out almost unconsciously or at least without effort. The patient in a moderately advanced stage when at rest will remain perfectly still without the numerous unconsidered movements that the normal person makes even in the state of relaxation. This sometimes gives the appearance of a person without any thoughts or emotions. Although there is a certain fatuousness and childishness about their mental attitude, there is very little real retardation of their intelligence. There are, of course, exceptions to this, as in Cases VIII. and XI. Thus, although intelligence and in some cases judgement are not seriously impaired, their behaviour in nearly all departs from the normal and their reactions to surroundings differ in each individual. It is this behaviour reaction that at times obscures the correct diagnosis. When the characteristic facies, bradykinesia and physical symptoms generally are slight and the subjective symptoms and behaviour generally are outside normal limits, the diagnosis of hysteria and neurasthenia, psychasthenia may be made or even a psychosis such as *dementia præcox* or *melancholia*. A diagnosis of mental deficiency may be made in children and young adults. Worster-Drought and Hardesty⁽¹⁵⁾ made observations upon a series of patients with regard to psycho-motor retardation in the presence of the post-encephalitis syndrome. Their conclusions were that the psycho-motor reaction time, that is the time between the giving of a simple order to a patient and the carrying of it out by the patient, was lengthened by about 50%. The cerebration time, however, is most appreciably altered. There is a change in the emotional reaction in these patients. Many of them are content and have a feeling of well-being and a lack of anxiety as to the nature of their illness. This has been pointed out by Gillespie.⁽¹⁷⁾ This mental placidity depends a

good deal upon the circumstances of the patient. The patient under observation in hospital where everything is easy for him, is usually placid and content. The patient presenting himself to the out-patients' department is often under difficult home circumstances and it is in these patients that the functional and hysterical symptoms are sometimes manifest. Golla⁽¹⁶⁾ has shown by means of the psycho-galvanometer that many forms of hysteria have their origin in the fact that the patient is underequipped emotionally. Emotion in this sense means the feeling tone possessed by a person which urges him to overcome the difficulties of life. This feeling tone has not normally its outlet in emotional expression, but according to the Lange James theory there must be a physical tendency to emotional influence before the feeling tone actually makes its appearance.

The bradykinetic patient lacks the physical ability towards emotional expression and so lacks the vital capacity to overcome both physical and psychological difficulties. Thus, in the face of ordinary difficulties this patient is liable to develop hysterical manifestations. These manifestations are easily removed by removing the immediate cause and differ from true hysteria in the lack of self-deception that is seen in the latter.

A factor no doubt in some of the functional symptoms met with in the sequelæ of encephalitis, is the knowledge of their physical inferiority as compared with the normal person. This leads to a psychological "inferiority complex." More especially is this seen in patients who have been thought to have been suffering merely from functional conditions and have been treated for such conditions. I have seen immediate improvement of these symptoms when the nature of the patient's affection is pointed out to him. In children there is apt to be a more serious mental deterioration. The child has not learnt self-control. The sense for picking up new impressions is lost, as the child's attention process is difficult to hold. The consequence is that a condition resembling a dementia arises. The child referred to above is negativistic and uncontrolled, but at the same time there is no true dementia. Her intelligence is fair, but it is impossible to make her learn to read or write. The condition seems to be more like a disuse atrophy of the higher faculties than a true dementia.

Prognosis.

These patients do not find their way into asylums according to the reports of English asylum authorities. Most of the patients who survive the primary stage of the disease and develop the first encephalitis syndrome, have been able to live at their homes. The deaths reported have usually been from intercurrent infections. The severity of the primary conditions seem to bear no proportion to that of the sequelæ. One may, however, say that sequelæ occur more frequently in the slighter primary cases owing to the insufficient treatment that is received in the early stages. Children are more prone to serious mental disturbances than adults.

Differential Diagnosis.

It may be illuminating to give a list of the conditions that in my own experience have been confused with the sequelæ of encephalitis.

1. *Paralysis agitans*.—The differentiation is extremely difficult at times; the points of difference have already been discussed.

2. Feeble-mindedness.—In young people especially the patient may be thought to be an ament, but ordinary conversation and intelligence tests do not substantiate this appearance.

3. The psychoses.—(a) *Dementia præcox* has to be taken into account because of the apparent katonía. There is, however, no sign of schyzophrenia or any of the ordinary signs of primary dementia, except perhaps in young people. There are times when the diagnosis may be difficult, but the points indicated previously will clear the way to the correct diagnosis.

(b) Melancholia may be difficult in some cases to differentiate from the sequelæ of encephalitis. The history and the cog-wheel rigidity may lend assistance in the diagnosis.

(c) Psycho-neurosis.—The fact that the patients exhibit symptoms of these conditions makes the diagnosis difficult. Neurasthenia and hysteria are the two conditions under this heading that may be mistaken.

(d) Neuro-syphilis may be simulated, especially when there are pupillary changes.

(e) Disseminated sclerosis.—Occasionally cranial nerve lesions together with other objective neurological findings may make the diagnosis difficult.

4. Neuritis.—I have seen two cases diagnosed as "neuritis" because of the pains and subjective sensations that some of these patients have in their limbs. In early cases sometimes the first sign of oncoming stiffness is a feeling of numbness and tingling. The reflexes are, however, normal. There may sometimes be slight objective secondary changes when the *thalamus opticus* is involved, but no peripheral anæsthesia.

5. Arthritis.—One case in my experience was diagnosed as an arthritis, because of the stiffness of the prominent part of the hip joint and a history of painful "joints." In the acute stages there is sometimes a history of acute pains in the limbs which the patients may say were in their "joints." On closer inquiry it will be found that the whole limb was tender to touch.

6. Spondylitis.—The stiffness of the back may give first indication of spondylitis.

7. Other neurological conditions.—Owing to the polymorphic distribution of the lesions, alteration of the deep reflexes with the extension reflex are occasionally found.

8. Internal secretion defect. I have lately seen patients who were treated with thyroid extract on the grounds that there was thyroid deficiency.

Treatment.

There is little to say about the treatment of this condition. On the physical side the muscular rigidity in many instances can be controlled by

hyoscine. Injections have the disadvantage of all long-continued treatment, besides giving only temporary relief. Moreover, some patients do not tolerate hyoscine given hyperdermically; one of these patients became confused after the injection and was at one time semiconscious for a brief period. Hyoscine hydrobromide by the mouth can be given more frequently and does not appear to have the same ill effects. In many cases this gives undoubted relief. I have seen no improvement with parathyroid gland therapy. Massage and reeducation at times will help. Psychotherapeutic methods cannot in removing, as much as trouble all worry and anxiety. It seems to be better to make the patient realize that he has physical disability and encourage him to overcome such disabilities by reeducation methods. Many of these patients have shed a number of their functional symptoms when the physical nature of their disease is pointed out to them.

Summary.

This paper is mainly a clinical survey of twelve cases of the sequelæ of lethargic encephalitis which have been under observation for varying periods of time.

The common features in all these cases is a more or less pronounced degree of the Parkinsonian syndrome.

The term "bradykinesia" includes the slowness of movement and absence of involuntary movements, facial expression, attitude and gait which is included in the Parkinsonian syndrome.

The muscle rigidity is to some extent independent of the bradykinesia, that is the two are not proportional to one another.

The typical tremor of *paralysis agitans* is not usually present, although many forms of tremor and involuntary movements have been described in this condition.

Mental symptoms are often present; most of these are of the so-called "functional" variety; actual psychoses are uncommon.

The anatomical basis of the bradykinetic syndrome is obscure. Kinnear Wilson does not think that the *corpus striatum* can be a centre to non-voluntary movements, but that the cerebral cortex is called into play in all movements which have in reality a conscious element.

Emotion is depressed; the facility for expression of emotion is part of the bradykinesia.

Treatment is mainly symptomatic once the diagnosis is established. Hyoscine hydrobromide by mouth appears to lessen rigidity in certain cases.

References.

- (1) T. S. Good: "Encephalitis Lethargica," *The Journal of Mental Science*, April, 1925, page 225.
- (2) A. Wimmer: "Chronic Epidemic Encephalitis," 1924.
- (3) A. G. Duncan: "The Sequelæ of Encephalitis Lethargica," *Brain*, Volume XLI., 1924, page 76.
- (4) Cruchet: "Paralysis Agitans and Encephalitis," *The Lancet*, August 8, 1925, page 205.
- (5) F. M. R. Walshe: "On Certain Tonic and Postural Reflexes in Hemiplegia," *Brain*, Volume XLVI., 1923, page 2.

(8) D. McAlpine: "The Pathology of the Parkinsonian Syndrome Following Encephalitis Lethargica, with Notes on the Occurrence of Calcification in this Disease," *Brain*, Volume XLI., 1923, page 255.

(9) C. S. Sherrington: "Integration of the Central Nervous System," page 308.

(10) A. de Kleijn: "Experimental Physiology of the Labyrinth," *Proceedings of the Royal Society of Medicine*, December, 1923, page 3.

(11) G. Riddoch and E. F. Buzzard: "Reflex Movements and Postural Reactions in Quadriplegia and Hemiplegia, with Especial Reference to those of the Upper Limb," *Brain*, Volume XLIV., 1921, page 397.

(12) F. M. R. Walshe: "On Variations in the Form of Reflex Movements, Notably the Babinski Plantar Movements, under Different Degrees of Spasticity and under the Influence of Magnus and de Kleijn's Tonis Neck Reflex," *Brain*, Volume XLVI., 1923, page 281.

(13) L. J. J. Muskens: "Central Connections of the Vestibular Nuclei," *Brain*, Volume XLV., 1924, page 452.

(14) G. H. Monrad-Krohn: "On the Dissociation of Voluntary and Emotional Innervation in Facial Paresis of Central Origin," *Brain*, Volume XLI., 1924, page 22.

(15) S. A. Kinnear Wilson: "Croonian Lecture," *The Lancet*, July 11, 1924, page 54, and August 8, 1924, page 278.

(16) J. I. Hunter: "The Sympathetic Innervation of Striated Muscle," *The British Medical Journal*, January 31, 1925, page 197. February 7, 1925, page 251. February 14, 1925, page 298. February 21, 1925, page 350. February 28, 1925, page 398.

(17) C. Worster-Drought and I. Hardcastle: "A Contribution to the Psycho-Pathology of Residual Encephalitis Lethargica," *Journal of Neurology and Psychopathology*, August, 1924, page 146.

(18) F. L. Golla: Croonian Lectures, 1922.

(19) R. D. Gillespie: "Epidemic Encephalitis, Some Psychical Sequelæ," *The Journal of Mental Science*, Volume LXX., 1924, page 1.

Reports of Cases.

PSEUDOMYXOMA PERITONEI.

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AND

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THE following clinical and pathological notes are furnished as a record of the origin of *pseudomyxoma peritonei* in a pathological state (mucocoele) of the vermiform appendix. The more usual mode of origin of pseudomyxoma is by the rupture of a mucinous cyst of the ovary and consequent implantation of mucin secreting cells on the peritoneum. The rarity of the condition about to be described and the necessity for distinguishing it from colloid carcinoma merit a detailed report.

Clinical History.

The patient, a man, aged forty-six years, consulted one of us (M.V.S.) during the last week of December, 1925, on account of pain which he located in the lower portion of the abdomen and chiefly on the right side. He complained also of nausea, but there had been no vomiting. He stated that he had suffered a severe attack of pain about three weeks before Christmas and again a fortnight later. He had not sought medical advice on either of these occasions, but had remained in bed for five days during the second attack.

Inquiry elicited the fact that for a period in 1912 the patient had been an inmate of the Greenvale Sanatorium under treatment for pulmonary tuberculosis. In 1924 he became ill with pneumonia and pleurisy and at that time was told he was tuberculous.

He had not lost in weight and was of good colour.

Physical examination of the chest failed to reveal any abnormality. Tenderness on palpation of the abdomen in the appendical region determined the diagnosis of appendicitis and operation was undertaken on January 12, 1926.

In the approach to the appendix through a "grid-iron" incision over McBurney's point the omentum was found spread over the presenting bowel to which it was adherent in several places. After the omentum had been separated from the bowel the appendix could be felt adhering to the posterior wall of the pelvis. It was gently liberated and when brought to the surface was found to be imbedded in a mass of plum-coloured jelly-like material. A piece of gelatinous substance of the size of a walnut was attached to the caecum.

The operation was completed by removal of the appendix and that portion of the caecum to which the pathological tissue was attached; no other deposits of "jelly" could be found in the pelvis or abdomen. Closure of the wound was effected without drainage.

It was natural that, pending pathological investigation, anxiety should be felt on account of the strong suggestion of colloid carcinoma conveyed by the findings, but the patient made an uninterrupted recovery and expressed himself as feeling perfectly well when seen three months after leaving hospital.

Pathological Examination and Comment.

The specimens as submitted for pathological examination consisted of the vermiform appendix and two pieces of gelatinous substance. To the serous coat of the appendix was attached a collection of jelly-like material, enveloping the viscus for approximately one-third of its circumference and measuring two centimetres in transverse section in its widest part.

The musculature of the appendix exhibited obvious pathological thickening, measuring 0.75 centimetre in transverse section.

The lumen was occupied by mucoid substance similar in appearance to that encircling the appendix, although there was no appreciable degree of distension of the viscus. This was no doubt precluded by the thickening and induration of the muscular coat.

The most interesting feature noted in the macroscopic inspection of the appendix is shown in the accompanying drawing, for which we are indebted to Dr. Frank L. Apperly. In the transverse section of the particular plane represented in the drawing the mucoid tissue is seen traversing the muscular coat, spreading under the serous coat and escaping into the peritoneal cavity through a perforation in the peritoneal investment of the appendix.

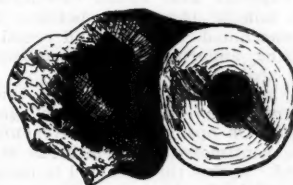


Figure Showing Transverse Section of *Pseudomyxoma Peritonei* Originating in the Vermiform Appendix.

In the microscopical sections the appearances were those of a chronic inflammatory process affecting the mucous membrane and musculature. The deeper layer of the tissue encircling the appendix, that is that next the peritoneum presented the histological characters of well vascularized granulation tissue in which the presence of many fibroblasts and young connective tissue cells indicated that the exudate was acquiring an organized attachment to the peritoneum. The occurrence of large numbers of eosinophile polymorphonuclear cells was a conspicuous feature. The peripheral portion of the section was almost structureless, a few elongated cells alone appearing.

The sections of the detached portions of gelatinous tissue showed this material to be devoid of cellular structure.

A reference to *pseudomyxoma peritonei*, resulting from the escape of mucinous material into the peritoneal cavity from rupture of the vermiform appendix is to be found

in Choyce's "System of Surgery," 1923, Volume I., page 581. The clinical features in a case recorded by Trotter are there described and they coincide to a remarkable degree with those of the patient who furnished the present example of *pseudomyxoma peritonei*.

In the case of Trotter's patient the diagnosis of colloid carcinoma was for a time maintained even after microscopical examination of a piece of omentum. The diagnosis from colloid carcinoma did not present such serious difficulty in the specimen above described, for although a strong suggestion of this variety of malignant disease was conveyed by naked eye inspection, it was in no way supported by the histological structure of the affected tissue.

A CASE OF PSEUDOMYXOMA PERITONEI OF APPENDICEAL ORIGIN IN A FEMALE.

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PSEUDOMYXOMA PERITONEI occurs not uncommonly in the female, but it almost invariably arises in connexion with an ovarian cystadenoma. The following case record presents several features of unusual interest.

Clinical History.

D.L., a saleswoman, aged thirty-five years, first sought advice on April 15, 1926. She stated that she had been losing weight for some time and that for fourteen days previously she had suffered from vague abdominal pain. For the two days immediately preceding the consultation she had been confined to bed with more severe pain which had become localized to the right iliac fossa. She had vomited three times on April 14 and repeatedly on April 15. There had been no definite rigor, but the patient had experienced a feeling of chill on April 14, since when she had felt feverish and thirsty. Her temperature was found to be 38.3° C., the pulse rate being 110 per minute. The bowels had been acting freely.

On physical examination the patient was observed to be a well developed young woman. The face was flushed and the abdomen, not distended, moved freely with respiration. A mass of the size of a cricket ball was visible in the right iliac fossa. Tenderness was elicited on palpation of the mass, which was situated between the umbilicus and the anterior superior iliac spine, two-thirds of it lying below a line joining these two points. No cutaneous hyperalgesia was noted. Vaginal and rectal examinations disclosed nothing abnormal and a provisional diagnosis of appendiceal abscess was made.

Anæsthesia was induced by ether and the mass approached through a short "grid-iron" incision in the abdominal wall. On opening the peritoneum at least two hundred cubic centimetres of gelatinous material flowed from the wound. It was then decided to open the abdomen in the mid-line in order to explore the general peritoneal cavity more effectively. The swelling discovered in the clinical examination was found to consist of the caecum, transverse colon and coils of small intestine welded into a mass which was much bigger than palpation had suggested. The whole was covered by myxomatous material and there were pockets of the same substance identical with that which escaped through the first incision, between the coils of bowel. All the pathological tissue was intensely vascular and bled freely on the slightest handling. The caecum and appendix were identified in the middle of the mass and it was estimated that the appendix was twenty centimetres (eight inches) in length and five centimetres (two inches) in diameter. The tubes and ovaries on both sides were normal in appearance.

Convalescence after the exploratory operation was uneventful.

During May, 1925, the patient received deep X ray therapy at the Alfred Hospital, two-thirds of an erythema dose being administered anteriorly and posteriorly, an interval of five days elapsing between the applications.

She was shown at a clinical meeting of the Victorian Branch of the British Medical Association held at the Women's Hospital in September last year. At that time she had gained ten kilograms in weight and the mass formerly present in the abdomen could not then be detected by physical examination. When seen on March 1, 1926, the patient appeared to be in perfect health and stated that for the previous six months she had been working at her regular occupation without any discomfort.

Comment.

The following points call for comment:

(i.) The extraordinary rarity of the condition. Eden and Lockyer mention three cases of pseudomyxoma of appendiceal origin in the female. In the male it is not so uncommon.

(ii.) Prognosis.—The prognosis of pseudomyxoma is generally bad. The process is not actually malignant, but the patient dies ultimately of obstruction or exhaustion. Frank⁽¹⁾ mentions a case in which one hundred and fifty-nine kilograms (three hundred and fifty pounds) of mucinous material were removed in twelve operations from one patient.

In the present instance it was impossible to have removed the whole mass operatively. To have removed the appendix was futile and would have exposed the patient to the grave risk of a faecal fistula. It is probable that no sutures would have held in the friable tissue.

At this date the result of treatment seems excellent. I know of one other case in which the patient was treated in a similar way three years ago and is still in good health. In that instance the condition followed the rupture of a pseudomyxomatous ovarian cyst. The pseudomyxomatous material is secreted by the cells of the adenomatous gland follicles and while it is easy to assume that these cells are sensitive to radiation, it is more difficult to understand how the material that has been poured out is absorbed. However, in the two cases that have come under my personal observation the results have been excellent.

Reference.

⁽¹⁾ R. T. Frank: "Gynaecological and Obstetric Pathology," 1922, page 393.

Reviews.

THE TREATMENT OF CHILDREN.

A NEW edition, the fourth, of Dr. John Thomson's "Clinical Study and Treatment of Sick Children" has appeared.¹ It contains several new articles dealing with the exudative diathesis, cephalic bruits, erythredema, arachnodactyly, pyknolepsy and hypertelorism, while the chapters on infantile scurvy, rickets, *encephalitis lethargica*, purpura, spastic paralysis, sporadic cretinism and the early treatment of mental defect have been revised and brought up to date. All that is new in the treatment of chronic hydrocephalus and congenital syphilis has also been incorporated. The author breaks new and valuable ground in the chapters on the home care of the deaf, blind and crippled. The book is mainly a clinical study of the diseases which are peculiar to children or which show characteristic differences, as they occur in early life, little being said about their pathology.

For the student and general practitioner the book should be invaluable and for the specialist there is much that is useful and new. Where detail is lacking, there are numerous references to other works.

The author, however, still appears to favour rather too prolonged medical treatment in congenital hypertrophy of the pylorus and in infectious diarrhoea makes no mention of the value of intraperitoneal injections of saline solution and of the injection of whole or of citrated blood in the treatment of severe cases of toxæmia and collapse in this disease.

¹ "The Clinical Study and Treatment of Sick Children," by John Thomson, M.D., LL.D., F.R.C.P. (Edinburgh); Fourth Edition, Rewritten and Enlarged; 1925. Edinburgh: Oliver and Boyd. Royal 8vo., pp. 919 with illustrations. Price: 30s. net.

The Medical Journal of Australia

SATURDAY, JUNE 19, 1926.

The Causation of Chronic Nephritis.

SINCE Richard Bright in 1827 called attention to the association of albuminuria and dropsy with pathological changes in the kidney, a large amount of energy has been expended in attempts to discover the underlying cause of nephritis. The structural changes found in the kidney were regarded as the alpha and omega of chronic nephritis. Attention was turned to the urine. The quantity of albumin was estimated and renal casts of various kinds were studied. The urea content of the urine was also estimated and useful information from a clinical standpoint was obtained. Later on with the advance of biological chemistry attention was focussed on the blood rather than on the urine. The same change was noticed in the attitude of investigators of the diabetes problem.

The blood is the medium for the conveyance of various substances required by the tissues for their metabolism. These substances do not appear in the urine unless their concentration exceeds a certain level or threshold. The threshold may be altered by disease or in order to compensate for disease elsewhere. The blood also conveys from the tissues materials which have been cast off as a result of their wear and tear. These waste products have no threshold and the percentage in the urine is determined by the concentrating power of the kidneys. Disease of the kidney may affect its concentrating power and biochemical tests may be of considerable assistance in determining the extent of the disease. Looked at from the functional rather than from the structural point of view chronic nephritis has been divided into hydræmic and azotæmic forms. The former is illustrated by parenchymatous and the latter by chronic interstitial nephritis. In the hydræmic type the threshold for both salt and water is raised and both are retained in the blood in

abnormal quantities. Nitrogen retention does not occur and the blood urea content is normal. In the azotæmic type the concentrating power of the kidney is damaged and there is a failure to eliminate solids. Nitrogen retention occurs and urea is found in the blood in increased amounts.

In a recent issue reference was made to work by Newburgh and Marsh and by Holding Anderson on protein diet in relation to chronic nephritis. Newburgh and Marsh have shown beyond question that when large amounts of certain amino acids are injected into the blood stream, they produce definite pathological changes in the kidney. The place of amino acids in the digestion of protein has been recognized of late years as the outcome of biochemical investigation. It was previously supposed that proteins became converted into peptones in the alimentary canal and were absorbed as such. It was taught that the product resulting from the earliest splitting of the original protein molecule could be absorbed without undergoing further hydrolysis. It is necessary to point out here that injection of protein into the blood causes a different state of affairs from that produced by ingestion of protein. When protein is injected a portion appears in the urine, in other words it is treated as a foreign constituent of the blood. At the same time there is some increase in the proteose-like substances of the blood and in the urea output. Hence it has been claimed that a limited amount of the injected protein may be used by the tissues. This utilization must be imperfect, because some of the protein appears in the urine. No protein appears in the urine of a healthy person after the ingestion of protein, save in very excessive quantities. After ingestion of protein hydrolysis occurs and the various amino acids are formed. These amino acids supply the nitrogenous needs of the body. As a matter of fact nitrogenous equilibrium has been maintained for long periods in animals fed with a diet containing no other source of nitrogen than the amino acids. If this feeding is undertaken, however, it is necessary to include in the diet all the amino acids which are formed by the normal hydrolysis of protein in the body. It has been shown that the excess of amino acid nitrogen occurring in the blood after a meal disappears with great rapidity

and that an increase of amino acids in the tissues takes place. If the supply of amino acids is too great for storage in the tissues, the chemical changes go a step further and the liver takes a hand. Ammonia is split off from the amino acid molecule and urea results. It can well be understood that when the tissues are replete with amino acids, the function of the liver might fail on account of some pathological condition and then the kidneys would be called upon to excrete unaltered amino acids. According to the work of Newburgh and Marsh such amino acids as arginin, aspartic acid, lysin, histidin, tyrosin, tryptophan and cystin would be calculated to inflict permanent injury on the renal cells if they were present in sufficient quantity. It is doubtful whether this happens in the human body under ordinary circumstances. It is also unlikely that the continued excretion of minute quantities of unaltered amino acid would produce permanent change in the kidney in the absence of another factor or factors. The frequent repetition of a small dose of morphine at relatively frequent intervals produces no permanent damage. Likewise the presence of lead in urine does not necessarily indicate a condition of lead poisoning; something more is necessary. The margin of safety provided by Nature in the metabolism of the human body is very wide and even when injury has been inflicted the power of recovery is enormous. Nevertheless the work of Newburgh and Marsh and of Holding Anderson and a host of others on the relationship of protein intake to structural change in the kidney is extremely important. It is for future workers to determine the degree of relevance which it bears to chronic nephritic change. Many avenues have not been exploited to the full. The exact significance of vascular disease in its relationship to nephritis has to be determined. Such questions as calcium metabolism, the rôles which chlorides and the hydrogen ion concentration of the blood play in the process have not been exhausted. There are probably other methods of investigation which have not been considered. The whole matter after all is one of the chemistry of the individual cell and of the discovery of conditions which induce abnormal forms of activity within its boundary.

Current Comment.

THE ÆTIOLOGY OF GASTRIC ULCER.

A VAST amount of work has been done in connexion with the elucidation of the mechanism of the production of gastric and duodenal ulcer. Much of this work has been carefully planned and skilfully applied. It has revealed many factors involved in the causation of the ulcers, although the various attempts to fix one particular agent have failed. Within recent times a change of attitude has become apparent in that several astute observers and original thinkers have set up the thesis that the ulcer, whether in the stomach or duodenum, is merely a local manifestation of a general bodily condition. Hypotheses have been evolved according to which the ulcers occur in persons who manifest vagotonic and sympathicotonic defects. While the ætiology of gastric and duodenal ulcer was sought in some special mechanism leading to superficial ulceration of the mucous membrane, little or no progress was made. The disease is not a local one and it is therefore futile to search for a chain of events leading to the local lesion unless the essential general defects are investigated at the same time. Dr. A. Winkelstein points out that ulcers occur in the gastric and duodenal mucosa of normal persons and that these ulcers undergo spontaneous healing.¹ This in itself is sufficient evidence that the disease depends on some special pathogenesis. Dr. Winkelstein also makes the suggestion that the main characteristic of the disease is the chronicity of the ulcerative process and the absence of all tendency to spontaneous healing. Moreover, experience of the process of healing indicates that certain mechanical factors are capable of delaying or preventing healing, such as constant irritation or narrowing of the vascular channels. In order to ascertain the nature of the bodily defects that inhibit the healing of the ulcers, he has studied a group of persons with gastric and duodenal ulcers and persons who have undergone surgical treatment on this account. If the local manifestations are dependent on some constitutional abnormality, the latter would persist after surgical treatment and should be recognizable. Surgical treatment may lead to the disappearance of the ulcers, but it cannot influence a pathological change that causes the ulcer to persist. Dr. Winkelstein conceived that the chronicity and lack of spontaneous healing of the ulcers might be due to a constitutional over-irritability. This, he postulated, could be produced as a result of an alkalosis, a parathyroid disturbance, the action of some toxic agent, a disturbance of the calcium metabolism, the presence of what is spoken of as a vegetative nervous system or some psychic influences. He admits that his investigations are as yet far from complete. He has tested the electrical irritability, the mechanical irritability and the response to adrenal extract and atropine and has found that persons with gastric or duodenal ulcer, including those who had been freed of the actual ulcer by surgical operation, responded much more readily than do normal people.

¹ *Archives of Internal Medicine*, April 15, 1926.

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¹ Surg

Thus, Chovstek's sign of mechanical irritability was elicited in twelve persons without evidence of ulcer, although they were all emaciated from other causes, out of a total of eighty persons. On the other hand sixty-four out of sixty-five persons with ulcer yielded the sign. None of the ulcer patients gave any indication of a vegetative nervous system, but seventeen out of nineteen patients with gastric neurosis presented all the characteristics. The calcium content of the blood of the ulcer patients was not ascertainably abnormal. Dr. Winkelstein emphasizes the fact that vagotonia and sympathicotonia as definite clinical states were not encountered among the persons with ulcer. He is inclined to the opinion that the presence of the qualities of these states may be used in the differential diagnosis between ulcer and gastric neurosis. The over-irritability observed in the ulcer patients and in the patients who had been subjected to operative treatment for ulcer, appears to be akin to a mild or latent form of tetany. He proposes to investigate the matter further. Unfortunately it is extremely difficult to determine the presence of a functional disturbance of the parathyroid gland.

The observations carried out by Dr. Winkelstein are capable of being controlled and extended by other clinicians. It would seem that the fundamental proposition that the persistence and failure of the healing processes of gastric and duodenal ulcer is dependent on some chemical or physical agent, is a rational one and is probably true. A condition of raised irritability of the gastric mucosa, however, seems to be rather too vague to explain the constitutional anomaly of the disease. It is conceivable that if the parathyroids are primarily at fault, the glandular imbalance may result in a modification of the normal hydrogen ion concentration of the secretion of the pyloric glands or of the duodenal secretion. A certain amount of information is available concerning the disturbance of the hormonal reaction in the stomach of persons with chronic ulceration of the stomach or duodenum. Hitherto the altered reaction of the secretion has been regarded as a possible cause of the ulcer, rather than as the chief factor in the prevention of the healing process. Too little is known concerning the physiological action of the parathyroids to admit at present of a rational method of restoring the defect. But it is along these lines that a further advance in knowledge may be anticipated.

PITUITRIN IN OBSTETRICS.

THE use of pituitary extract during the progress of labour has become very common. Occasionally warnings have been issued on the ground that rupture of the uterus is liable to occur if undue obstruction to the expulsion of the foetus is present. It matters not whether this obstruction is due to non-dilatation of the *os uteri* or to narrowness of the pelvic outlet. An instructive case of rupture of the uterus following the administration of pituitary extract has recently been reported by Dr. J. Garland Sherrill.¹ The patient, a healthy young woman,

was pregnant for the first time. Some uterine inertia was present, delivery was effected by means of instruments and manual extraction of the placenta was necessary. During the progress of labour three doses of obstetrical pituitary extract, each 0.5 cubic centimetre, were given at intervals of two hours. No information is given as to the relationship of the application of forceps to the administration of the drug. After removal of the placenta serious hæmorrhage occurred and the patient became collapsed. Sepsis occurred and after thirty days during which a uterine irrigation was carried out, abdominal section was performed. Two small tears were found in the peritoneal covering of the uterus and these were connected in each instance with wide separation of the muscular and mucous coats. Dr. Sherrill points out that the interesting features of the case included the failure of hæmorrhage and collapse to arouse any suspicion of the occurrence of rupture. In addition no peritonitis was found as a result of the injury. The lessons to be learned from this case are that if pituitary extract is used, the obstetrician should suspect rupture of the uterus when hæmorrhage and collapse occur. Moreover, he should not be satisfied until inspection has revealed the source of the hæmorrhage and settled the diagnosis.

A MEDICAL INSURANCE COMPANY.

THE Victorian Branch of the British Medical Association has recently instituted an insurance company for the benefit of its members. The company is known as the British Medical Insurance Company of Victoria, Limited. It will operate under the mutual system which means that the policy holders and the Victorian Branch will divide the profits. It is the intention of the Directors to reinsure the whole of the business with another insurance company, named the Automobile Insurance Company of Australia, Limited, from which a substantial commission will be received. This so-called exchange commission will provide funds for a 10% bonus to be paid on all policies taken out by the members of the Victorian Branch with the new company. It is claimed that the moral hazard of insurance business with medical practitioners is excellent and in consequence greater benefits will accrue.

Members transferring their policies to the British Medical Insurance Company of Victoria will not lose any bonus due or accruing to them under any personal accident and sickness or motor car policy contracts. A comprehensive motor car policy will be issued to members of the Victorian Branch of the British Medical Association. This policy offers many advantages not available in connexion with other motor car policies in Victoria. The premium will be lower than that charged by other companies. Members of the Victorian Branch are advised to transfer their insurance policies to the new company. The Directors are Dr. C. H. Mollison, Dr. J. Newman Morris, Dr. R. H. Fetherston and Dr. W. Kent Hughes. An experienced underwriter is acting for the company.

¹ *Surgery, Gynecology and Obstetrics*, May, 1926.

Abstracts from Current Medical Literature.

BACTERIOLOGY AND IMMUNOLOGY.

Bacterial Flora and the Hydrogen Ion Concentration of the Duodenum.

LLOYD ARNOLD AND LOUIS BRODY (*The Journal of Infectious Diseases*, March, 1926) have investigated the bacterial flora of the duodenum and the effect produced by alteration of the hydrogen ion concentration. The contents of the duodenum and of the greater part of the jejunum are almost free of bacteria during the post-digestive period. The microorganisms usually found comprise Gram-positive cocci, staphylococci and enterococci. Sometimes isolated Gram-negative bacilli are found. McClendon and his collaborators found that the pH of the contents of the duodenum of healthy men ranged from 4.1 to 6.5. The period of their observations was five days. Some observers find a variation of from pH 4 to pH 8. The authors regard pH 4 as representing a recent outpouring of gastric contents and pH 8 as being due to a recent secretion from the pancreas not mixed with gastric contents. They add that most of the results are between pH 5 and pH 6.5. They mention some of the factors which influence the hydrogen ion concentration of the duodenal contents. The first of these is the gastric contents; by this is meant the total and free acidity of the contents. Another factor comprises the secretions from the pancreas. These are alkaline in reaction and are probably continuous with increased amount of secretion when digestible material is present in the duodenum. A third factor consists of the alkaline secretions of the liver. These are not so well buffered as are the pancreatic secretions. Fourthly the secretions from the mucosa of the duodenum are reported to be alkaline in reaction, but their composition is as yet unknown. The authors carried out a series of controlled experiments. They found that ligation of the pancreatic ducts does not lead to a change in the bacterial flora of the duodenum. Carlson has shown that elevation of the body temperature of the dog is accompanied by a depression of gastric secretion. The authors caused a rise in temperature by the injection of staphylococcus vaccine. They found that the hydrogen ion concentration changed from neutral to slightly alkaline (pH 6.8 to pH 7.4) and that bacterial flora became the same as that found in the ileum and caecum. When the temperature became normal, the duodenal flora and the hydrogen ion concentration returned to normal. When alkaline phosphate buffer solution (pH 8.9) was injected directly into the duodenum of a normal dog the bacterial flora changed from the type usually found in the duodenum to that characteristic of the ileum and caecum. When acid buffer solution

(pH 2.6) was injected into the duodenum the bacterial flora did not change. In most instances it was found that a leucopenia accompanied acid injections and that in every instance except two leucocytosis was the rule after alkaline injections. When the duodenal contents were alkaline either as a result of gastric secretory disturbances or the injection into the duodenum of alkaline buffer solutions the flora was that of the ileum and colon. The injection of an acid buffer solution in these circumstances did not correct this state of affairs. The acid solution stimulated peristalsis and was soon passed out of the small intestine. The addition of an acid solution, however, caused readjustment to take place sooner. The authors in their conclusion state that the maintenance of the normal hydrogen ion concentration of the contents of the duodenum is dependent to a great extent on normal gastric secretory function.

The Soluble Constituents of Pneumococci.

LLOYD D. FELTON AND G. HOWARD BAILEY (*The Journal of Infectious Diseases*, February, 1926) have sought the explanation of the phenomenon of the failure of large doses of anti-pneumococcal serum to protect mice from lethal quantities of pneumococci while smaller doses afford complete protection. They have reason to believe that this is caused by the presence of two substances in the serum, one a protective agent and the other a substance antagonistic to the animal's defence. Other investigators have found that a specific substance can be extracted from pneumococci in aqueous solution which acts like an aggrassin. The authors have studied this water soluble specific substance both *in vitro* and *in vivo*. In the first place they found that mice died readily when a relatively avirulent strain of pneumococci was injected together with the soluble substance. In the next place they precipitated a high potency Type II. serum with soluble substance in varying concentration. The greatest amount of precipitation occurred with a concentration of one part in two thousand parts. This precipitate displayed no protection and the supernatant fluid also failed to protect. On the other hand when the soluble substance in a concentration of one part in twenty thousand was used, the precipitate afforded approximately the same protection as the original serum, while the supernatant fluid was equally protective. The soluble substance itself proved to be non-toxic. They adduce considerable argument in favour of the hypothesis that the soluble specific substance acts in a manner similar to that postulated by Rosenow and called by him "virulin." They conducted a large number of neutralization experiments both with antipneumococcal serum and antibody solution. They have demonstrated the possibility of dissociation and liberation of the protective protein by heat. They

also studied the phenomenon of precipitation under varying conditions.

In a second article published in the same issue the authors give an account of numerous experiments undertaken to disclose the nature of the precipitates referred to above. All the protective substance of Type II. anti-pneumococcal serum can be precipitated with appropriate amounts of the soluble specific substance. The precipitate is insoluble in water, sparingly soluble in 3% sodium chloride solution and very soluble in dilute acid or alkali when the salt content is low. The hydrogen ion concentration of a solution of the specific precipitate that proved to be the weakest solution capable of exerting protection, was found to be equivalent to pH 6.6. They carried out some experiments that led them to conclude that the protein of the protective substance is not of the nature of eglobulin. Assuming that this protein has the same molecular weight as pseudoglobulin, the combining ratio per weight of soluble specific substance was found to be as one to fifteen. The molecular weight of the soluble substance was calculated to be of the order of 5,300 and the total molecular weight of the resulting combination 85,300. They ascertained that the specific precipitate was readily dissociated by means of dilute acid or alkaline solutions. After dissociation the solution exercised no protective action. The protective substance, however, was thrown out of solution by the addition to the acid solution of sufficient sodium chloride to produce quarter saturation. In a similar manner the precipitate was dissolved by means of sodium carbonate, disodium phosphate, saccharose or sodium chloride. The addition of sodium sulphate sufficient to produce a solution of 18% produced a precipitate of nearly all the protective substance. In association with the protective substance thus thrown out of solution some antagonistic substance was found.

The Absorption of Undissociated Protein.

MATTHEW WALZER has endeavoured to demonstrate the absorption of incompletely digested protein in healthy human beings (*The Journal of Immunology*, April, 1926). It has been found that the injection into the skin of the serum of a person rendered hypersensitive to an antigen induces a local sensitization to the same substance. He discovered two children who were hypersensitive to several articles of food. About fifty grammes of raw herring was taken by a normal person by mouth. The serum of one child sensitive to fish was then applied intradermally. A reaction appeared in ten to twenty minutes after the meal. It lasted for a half to two hours and then faded in gradual stages. The reaction to raw egg appeared after from thirty to one hundred minutes. The test was applied to fifty subjects and failed in six. The author is investigating the cause of the failures.

He claims that the method is simple, rapid and specific and definitely proves the absorption of partially digested protein.

HYGIENE.

Industrial Intoxication by Lead, Benzol and Nickel.

NORMAN GWYN (*The Canadian Medical Association Journal*, March, 1926) holds that even in the absence of the typical symptoms an indisposed worker should be carefully examined for signs of lead absorption, if he is engaged in an occupation involving use of lead in any form whatever. Workers subjected to the risk of absorbing, ingesting or inhaling lead, its salts and its fumes, may manifest the following large range of symptoms and signs long before the typical picture of intoxication has developed and such symptoms suggest that lead is being absorbed. These symptoms include loss of strength and weight, failing or double vision, ringing in the ears or deafness, nausea, anorexia, abdominal distension, mild colic, diseased gums and also incontinence of urine, palpitation and nose bleeding, neuralgia, weakness in the legs, headache, vertigo, syncope, depression and failing memory. Careful examination for a blue line, for stippled red cells in the blood, for traces of lead in the urine and faeces is essential in the face of such premonitory symptoms. In the very acute and pronounced cases there is usually no difficulty in determining the nature of the disorder. Milder degrees of intoxication require for their detection and explanation an exact appreciation of the nature of a man's work, a knowledge of the fact that lead is stored in the bones and may remain there till conditions of ill-health or an acidosis may call it forth and finally a recognition of the existence of the host of minor but incapacitating symptoms. Benzol is widely used as a solvent, a vehicle or as a motor fuel. It is used as a solvent in the rubber industry, in the manufacture of artificial leather, certain paints, floor waxes, varnish removers and certain kinds of cement. In a worker exposed to the fumes of benzol and presenting signs and symptoms resembling *purpura hemorrhagica* or pernicious anemia benzol poisoning must be thought of. The diagnosis depends on a history of exposure to the fumes of benzol, a history of failing strength, drowsiness, anorexia and vomiting with mental depression, pallor, breathlessness and bleeding from the nose and gums. Moreover, the examination reveals a pallid, weakened man with signs of recent or old hemorrhages, with a low blood count, a low hemoglobin and low white cell count with other signs of anemia such as poikilocytosis and variations in the size of the red cells to both above and below normal. Nickel poisoning takes the form of a rash occurring on the skin. It is most apt to develop in hot weather, seldom in winter. The rash first appears on exposed surfaces or where at any point

an irritation exists such as may arise from stiff collars or cuffs or in the folds of the skin at the joints *et cetera*. The rash is characterized by an intense itching, worse at night. This itching is followed in a short time by the appearance of a papular pin-point rash which may later become wet and eczematous or may be eczematous from the first. From a small beginning the rash is apt to spread rapidly to neighbouring areas on the skin.

Notification of Occupational Diseases.

O. A. CANNON (*The Canadian Medical Association Journal*, March, 1926) points out that industrial hygiene is one of the major branches in the field of preventive medicine and that occupational diseases can be brought under control and very often eliminated from industry. He cites two instances. The Pullman Car Company had seventy-seven cases of lead poisoning in one year, but found that the introduction of supervised washing of the hands enabled them to report no case the following year. A Paris green factory in London lost through sickness due to arsenic dust, twenty-five men a year, but found that the installation of suitable apparatus to eliminate the dust resulted in an absence of cases the following year. He adds that it is doubtful if there is any occupational disease which would not yield to a knowledge of the manufacturing process, the chemistry of the substances and the effects of these substances on the human organism. With compulsory notification in operation the report to a local medical officer of health that a case of industrial disease had occurred in a workman employed in a certain industry would be a signal for an investigation at that industry with the object of preventing other workmen from being affected by the same condition. Furthermore manufacturing processes are continually changing, new products being turned out and new methods being devised and all are accompanied by new hazards to the workman. Facilities must therefore be provided for the detection and prevention of the new hazards arising in the course of industrial development. If, however, all cases were reported to a central authority with the clinical signs and symptoms there would soon be piled up for analysis a volume of experience which would be of great value in the diagnosis, treatment and prevention of these diseases. The author suggests that every medical practitioner attending on or called in to visit a patient whom he believes to be suffering from anthrax, lead poisoning, mercurial poisoning, phosphorus poisoning, arsenical poisoning, anchylostomiasis, miner's phthisis, benzol poisoning, silicosis or any other disease believed by the medical practitioner to be due to the occupation of the patient, shall immediately give notice on the form provided to the medical officer of health of the municipality in which the patient is employed. The practice of listing certain diseases and stopping

there is not to be recommended as it closes the door to conditions not listed and would defeat the purpose for which notification is desired, namely the study of the indefinite conditions. Therefore the clause should be framed so that even those industrial illnesses to which the doctors could not assign a name, would nevertheless be reported.

The Industrial Medical Practitioner.

W. IRVING CLARK (*The Canadian Medical Association Journal*, March, 1926) describes the medical practitioner in industry as one who applies the principles of modern medicine and surgery to the industrial worker, ill or well, supplementing the remedial agencies of medicine by the sound application of hygiene, sanitation and accident prevention and who in addition has an adequate and cooperative appreciation of the social, economic and administrative problems and responsibilities of industry in its relation to society. He then points out how great has been the growth of manufactures in the last twenty years and how the grouping of large numbers of men in factories has produced changes in health conditions due to unhygienic surroundings and also introduced the particular hazards which always exist in industry as a whole. It soon became evident to the manufacturer that a definite responsibility for the safety and health of his employees was his and he soon realized that there was an economic principle present as well, for if employees were losing time because of illness and accident and because of wrong hygienic conditions, from a purely economic point of view it was to his advantage to do something in the way of prevention. The author then points out that the medical practitioner in industry must have a good grounding in general medicine, must be a good surgeon, having had operating room experience in a first-class hospital and must also have a general knowledge of public health problems. In addition he should be the type of man who cooperates with others and who is willing to sink his own personality in team work when such action is needed. He must know the factory in which he works, must know manufacturing processes, must know the working conditions of every job in his factory and above all must know the workman at his work. Because of his responsibility for the health and safety of the employees in the plant, the industrial medical practitioner should be subordinate to no other individual than a man holding the highest position. Unless he has the sympathetic understanding of the head of the factory his influence will be weakened and his ability to spread his work throughout the organization will be materially diminished. In conclusion the writer discusses the industrial physician in the community. His influence in the community is great. The employer is very much a human being and will listen to a medical practitioner, while he may not heed legislation.

British Medical Association News.

ANNUAL MEETING.

THE ANNUAL MEETING OF THE WESTERN AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Claremont Hospital for the Insane on March 21, 1926, Dr. FERGUSON STEWART, the President, in the chair.

President's Address.

Dr. FERGUSON STEWART delivered an address entitled: "The Relation of the British Medical Association to State Craft" (see page 689). He also read the annual report. The report is in the following terms:

Annual Report of the Council.

Membership of Branch.

The membership of this Branch has increased during the year from 180 to 191, being over 80% of the resident registered practitioners.

It is with much regret that I have to report the death during the year of Dr. R. C. Merryweather. Letters of condolence were forwarded to his widow. Dr. Merryweather was an active member of the Branch for many years, having occupied the position of President in 1915, London representative in 1920 and was a member of the Council for six years. His premature death was a great loss to the Association.

Meetings.

The Council held nine meetings with an average attendance of seven. There were nine general meetings with an average attendance of twenty-seven and during the year the Ethical Committee held four meetings.

A successful annual dinner was held this year at the Palace Hotel when forty-three members were present, Dr. William Trethowan being the guest of honour of the evening.

Clinical Evenings.

During the year three clinical evenings were held, two at the Perth Public Hospital kindly arranged by Dr. Mackenzie and another at the Children's Hospital, kindly arranged by Dr. Hislop.

During the year interesting cases were reported on by Drs. Beveridge, Couch, Crisp, East, F. Gill, H. B. Gill, Gray, Harris, Hislop, Holland, Joyce, Juett, Langley, Lotz, Maitland, McConnell, MacGlashan, Mackenzie, McWhae, Nelson, Paton, Robertson and White.

Papers.

During the year much interest was taken in the reading and discussion of papers. Papers were read by the following members: Drs. Blackall, Hislop, Holland, Syme Johnson, G. Mayrhofer, MacGlashan and Stewart.

Council Work.

The work of the Council during the year has been very heavy and many matters of importance have been dealt with and advice and assistance given to members in reference to agreements, subsidies *et cetera*. I desire to mention a few of the most important matters.

Library.

During the year the Council made arrangements with the Board of the Perth Hospital who kindly provided space in the boardroom for a British Medical Association Library. Arrangements have been made for the purchase and binding of periodicals and books and the library has been placed under the control of two honorary librarians, Dr. F. J. Clark and Dr. Troup. It is hoped that an annual grant will be made by the Association to maintain this useful work.

King Edward Memorial Hospital.

A subcommittee consisting of Drs. Clark and Hislop have conferred with the Principal Medical Officer of the Health Department and it has been arranged to appoint one resident.

Hospital Abuses.

A considerable time was spent by your subcommittee inquiring into this matter as it affected the public hospitals in the metropolitan area and a lengthy report will be submitted for your consideration at the April meeting.

Workers' Compensation Act.

Much consideration has been given to this matter by the Council. A request was made to the Accident Underwriters' Association that all doctors' accounts should be paid direct and intimated to the Underwriters' Association that the Council was prepared to appoint a subcommittee to report on any doctors' accounts, considered unreasonable by the insurance companies. This request and offer has not been definitely accepted yet by the Accident Underwriters' Association.

Legal advice has been obtained from the solicitors of the Branch for the purpose of excluding injured workers entitled to compensation under the *Workers' Compensation Act* from benefits under the Model Lodge Agreement, the Timber Mill Agreement and all other agreements. The legal advice we have received is to the effect that injured workers entitled to compensation under the *Workers' Compensation Act* must be specially excluded in all agreements, otherwise the medical practitioner entering into such agreements cannot claim medical fees under the *Workers' Compensation Act*, as such expenses are not incurred by the workers. It is clear that the effect of this is that the insurance companies are saving paying medical expenses for which they receive insurance premiums. Already the matter of such injured workers has been definitely referred to the Friendly Societies' Council and the Timber Mill Agreements Committee.

Australasian Medical Publishing Company, Limited.

During the year the Council recommended that Dr. D. D. Paton should take the place of the late Dr. Merryweather as member of this Company.

Dr. Penfold's Lecture.

During the year a very valuable lecture was given in Perth by Dr. Penfold, of the Commonwealth Laboratory, Melbourne, on "Serums and Vaccines" and arrangements were made by the Council to have this printed in pamphlet form and issued to every medical practitioner throughout the State. On the approval of the Minister this paper was kindly printed by the Government Printer free of charge and was issued to every medical practitioner in the State and also to the Western Australian Branch of the Australian Veterinary Association.

Alteration of Rules.

In the early part of the year Rule 38 was altered to give the Council power to appoint the Principal Medical Officer of the Health Department a member of the Council provided he was a member of the Association.

The alteration of Ethical Rule 62a has had the careful consideration of the Council. The proposed alteration will be submitted to you tonight for ratification. It is to the effect that one notice may be published in the paper of the resumption of private practice after a week's absence. The object of such advertisement is to give the patients notice that the doctor has resumed practice and I hope you will approve of such alteration.

Opening of New London Office.

Dr. Morlet who visited England, was asked to attend the opening of the new London office and represent the Branch at this ceremony.

Visit of Dr. W. N. Robertson.

The Federal representative for Australia at the new London office passed through Fremantle early in the year on his way to London and was entertained by the President and the Federal representatives. Through Dr. Robertson, expressions of goodwill from this Branch were forwarded to the parent body in London.

Annual Meeting at Bath, 1925.

Drs. Morlet and Tregonning both of whom were visiting England, were asked to attend this meeting to represent this Branch.

Annual Meeting at Nottingham.

The annual meeting this year will be held at Nottingham and an invitation from the President Elect has been issued to members of this Branch. It is desirable to know any members who are going to England this year, so that arrangements can be made for delegates to this meeting. The Council has again recommended the appointment of Dr. T. P. Dunhill to the representative body of the Association for 1926-1927.

Australasian Association for the Advancement of Science.

Australasian Association for the Advancement of Science will hold the meeting this year in Perth and the Branch has been asked to support this meeting. The next meeting cannot be held in Perth until after an interval of fifteen years and it is therefore hoped to make the Perth meeting successful.

Election of Office Bearers.

Dr. Ferguson Stewart, the President, said that 191 nominations had been issued on February 2 and only six had been returned.

Dr. Dixie Clement, who was retiring as ex-President from the Council, had for many years taken the keenest interest in the Branch. Dr. Clement had a long record of energetic and useful work; he had been Honorary Secretary for three years (1908-1910), Member of Council for six years (1914-1919), Vice-President (1923), President (1924) and ex-President the previous year. He had also been six years a member of the Ethical Committee (1915-1920).

Dr. Stewart took the opportunity on behalf of the Council who had worked with Dr. Clement in such close and friendly relations and in the Branch as a whole, of thanking him for his past valuable services.

There was no election, Dr. Gibson having been proposed and seconded for the position of Vice-President and Dr. J. J. Holland being proposed and seconded for the position of Member of the Council. One other member had been proposed as Vice-President and one other member as member of the Council but as their nominations had not been seconded they could not stand for election.

He declared the following office bearers for 1926 duly elected:

President: Dr. T. L. Anderson.

Vice-President: Dr. A. H. Gibson.

Ex-President: Dr. Ferguson Stewart.

Honorary Treasurer: Dr. D. D. Paton.

Honorary Secretary: Dr. R. H. Crisp.

Members of the Council: Dr. E. C. East, Dr. R. S.

McGregor, Dr. J. J. Holland, the Principal Medical Officer of the Health Department (Dr. R. C. E. Atkinson), by special resolution.

Members of the Ethical Committee: Dr. G. W. Barker, Dr. H. B. Gill, Dr. W. H. Nelson.

Financial Statement.

The financial statement for the year 1925-1926 was presented by the Honorary Treasurer, Dr. D. D. Paton, and adopted.

Expression of Thanks.

Dr. Ferguson Stewart thanked Dr. J. T. Anderson, Inspector-General for the Insane, on behalf of the Branch for his great kindness in allowing the Branch to hold the annual meeting at Claremont and also for entertaining the members at tea before the meeting.

On rising to respond to the thanks of the meeting, conveyed to him through the President, Dr. J. T. ANDERSON said these gatherings had always been a pleasure to him. It was the last time that he would have the pleasure of inviting them, as he was retiring from professional activities. The custom of having their company at dinner

once a year and of holding the annual general meeting at the Hospital had commenced with his predecessor and he felt that it would give his successor as much pleasure as it had given him.

Induction of President.

Dr. Ferguson Stewart thanked the members of the Branch and especially the members of the Council for their loyal support during his year of office as President.

He then vacated the chair in favour of Dr. T. L. Anderson.

Election of Auditors.

Dr. A. E. Randell and Dr. E. Clapperton Dean were unanimously reappointed Honorary Auditors.

Annual General Meeting at Bath.

Dr. Tregonning gave an interesting report on the business discussed at the annual general meeting held at Bath and particulars of social events *et cetera* which were much enjoyed.

Librarian's Report.

Librarian's report was read by Dr. F. J. Clark. The list of periodicals ordered was read, Dr. Clark stating that the first year's journals were somewhat in the nature of an experiment.

Rule re Resumption of Practice.

On the motion of Dr. T. L. Anderson, seconded by Dr. Crisp, it was resolved to add the following words to Rule 62A (4):

Where practitioners resume practice after an absence of not less than one week, they may notify their patients by advertisement in the Press on one day only.

SCIENTIFIC.

At the annual meeting of the Western Australian Branch of the British Medical Association held at the Hospital for the Insane, Claremont, Western Australia, Dr. H. W. Moxon read a paper entitled: "Cerebral Vascular Lesions: Their Varieties, Symptoms and Sequelae" (see page 690).

Dr. T. L. ANDERSON thanked Dr. Moxon for his paper which had interested him intensely.

Dr. J. J. HOLLAND said that Dr. Moxon's views on the lessening of blood pressure were very enlightening. In his opinion more trouble than help had been received from the sphygmomanometer. The profession had been to blame largely, because they would tell their patients what blood pressure reading they found. Many people were unduly worried because of the knowledge that they had a high blood pressure. There was an evil influence in the doctrine of lower blood pressures and he thought Dr. Moxon deserved the thanks of the Branch for his lucid explanation.

Dr. N. M. CUTHBERT stated that he had been somewhat staggered some short time previously at reading in Price's textbook of medicine of the stimulatory treatment of cerebral hæmorrhage. On thought and after listening to Dr. Moxon the explanation, however, was clear. Stimulation was administered to keep up the cerebral circulation. It was apparent that the general symptoms of apoplexy were the result of cerebral ischæmia produced by the raised intracranial pressure either from hæmorrhage or oedema and that nature attempted to combat this ischæmia by a reflex raising of the blood pressure, to keep the cerebral circulation going. Stimulation would help Nature to keep the circulation active.

Dr. K. MOSS said that if stimulation was good in cerebral hæmorrhage, it ought to be good before. Would Dr. Moxon recommend stimulation in a patient with a blood pressure of two hundred millimetres of mercury, who had had no hæmorrhage.

Dr. D. M. McWHAE said that he wished to compliment Dr. Moxon on the very great skill and ability with which he had put his paper before the members. An extraordinary amount of ground had been covered and yet

all had been made perfectly lucid and there was little left on which he could comment. Dr. McWhae said that the diagnosis between cerebral hemorrhage and cerebral thrombosis was often exceedingly difficult. The only way to be sure lay in resorting to a lumbar puncture and this had also been recommended as a method of treatment.

Dr. A. W. Farmer asked Dr. Moxon what the blood pressure was in cases of hemorrhage around the circle of Willis.

Dr. Moxon in reply stated that he would do his best to answer some of the questions which had been raised, but some of them naturally enough involved the question of hyperplasia rather than the subject under discussion.

If the patient has a cerebral vascular lesion such as atheroma and at the same time a low blood pressure, an attempt should be made to raise the blood pressure to overcome the possibility of thrombosis. Dr. Moxon instanced a case of a man of forty-eight with very thick arteries, but a systolic blood pressure of one hundred and forty-six millimetres of mercury. The ophthalmologist had reported sclerosis of ophthalmic arteries. Dr. Moxon prescribed treatment in the form of citrate of potash, tincture of digitalis and *liquor strychninae*.

In a recent hemorrhage the symptoms and signs were derived from an obstructed circulation and as the intracranial pressure increased, Nature attempted to raise the blood pressure to carry on the circulation.

Dr. Moxon, in conclusion, thanked the members present for the interest they had displayed in the subject and for the kind words they had spoken of him.

Dr. J. G. Hislop said that he could not let the evening close without concurring with the views expressed by the President in his address. He felt that the medical profession had kept itself aloof from the attempts which were being made to educate the public in matters of medical interest. There was no doubt that the public were looking to the profession to take a lead in progress of preventive medicine, but so far in vain. So long as they held aloof the Press would get what they could of medical interest. It would be preferable if comments bore the authority of the Branch and were not merely the unsigned expressions of opinion of individuals.

Dr. Hislop hoped that the Council would take up the subject of Dr. Stewart's address and formulate some scheme.

Medical Societies.

THE SYDNEY HOSPITAL CLINICAL SOCIETY.

A MEETING OF THE SYDNEY HOSPITAL CLINICAL SOCIETY was held at the Maitland Lecture Theatre, Sydney Hospital, on March 20, 1926, Dr. RALPH WORRELL in the chair.

Xanthoma Tuberosum Multiplex.

Dr. E. H. STOKES showed a female patient, aged fifty-four years, married, whose occupation was confined to domestic duties. He said that he was showing the patient for two reasons, firstly because of the rarity of the condition and secondly in order to invite suggestion as to further treatment.

In January, 1925, the patient had noticed the appearance of yellow streaks in the creases of the palms of her two hands, a phenomenon attributed by a masseuse to an increase in acid in the blood. In June, 1925, several yellow plaques (xanthomata) had appeared on both elbows. In October, 1925, she had suffered from considerable worry and anxiety as her husband had just undergone a serious operation. On November 7, 1925, she had been seized with giddiness followed by staggering and by buzzing in the ears. Two days later she had vomited several times and then became deaf in the left ear. The deafness had been of the labyrinthine type.

In January, 1926, a crop of xanthoma nodules had appeared in various parts of the body. Both shoulders and the posterior aspect of both arms had been affected. The

creases of the palms of both her hands had been filled with yellow material and xanthoma nodules had been present in the flexures of the fingers. Both buttocks, the anterior aspects of both knees, the calf of the right leg, both heels and the dorsal aspects of both feet had also been covered with xanthoma nodules. The parts subjected to pressure had appeared especially affected. The skin of the head and neck of the thorax and abdomen as well as the mucous membrane of the mouth had been unaffected.

Apart from the neurasthenic condition associated with the worry about her husband's operation and aggravated by the menopause she had not suffered from any illness nor had she undergone any major operation. She had not suffered from gall stones.

The patient's general appearance had suggested hypothyroidism. Her skin had been dry and the outer third of her eyebrows had been scanty. The heart had been normal and the arteries not thickened. The systolic blood pressure had been 136 millimetres and the diastolic blood pressure 100 millimetres of mercury. The lungs had appeared normal and the liver and spleen had both been normal in regard to size. The urine had been acid in reaction with specific gravity of 1020 and it had contained neither albumin nor sugar. It had been tested on several occasions. The ocular fundi had been normal. Her mouth had contained a large number of devitalized teeth. Skiagrams taken by Dr. J. G. Edwards had shown that several of these teeth were affected by septic processes.

A nodule had been excised from the right elbow and another from the right heel. Sections of these nodules had been examined by Dr. Inglis and Dr. Shearman who had reported that the histological appearances were typical of xanthoma.

A glucose tolerance test had been performed by Dr. Shearman on February 8, 1926, with the following result: (i.) Before giving fifty grammes of glucose, blood sugar = 0.7% with no sugar in the urine; (ii.) one hour after giving fifty grammes of glucose, blood sugar = 0.15% with no sugar in the urine; (iii.) two hours after giving fifty grammes of glucose, blood sugar = 0.15% with no sugar in the urine. There had thus been no serious interference with the carbohydrate metabolism.

The cholesterol content of the blood had been estimated on February 12, 1926, and had been found, as was usual in xanthomatosis, to be considerably higher than normal. The figure had been three hundred and twenty milligrammes per hundred cubic centimetres, the normal amount being one hundred and forty to one hundred and eighty milligrammes. It had been noticed that the blood serum contained a considerable quantity of lipid. These findings had indicated that there was a serious defect in the fat metabolism of the body.

On April 16, 1926, the basal metabolic rate had been estimated by Dr. Hansman and had been found to be -16%. Dr. Hansman had also found that neither the direct nor indirect reaction was obtained from the Van den Bergh test. The serum had not reacted to the Wassermann test.

Dr. Langlois Johnston had seen the patient in consultation and had confirmed the diagnosis, he had agreed that the essential defect was evidently in the metabolism of the fats. Acting upon the suggestion of Dr. Harold Ritchie, he had given the patient 0.06 gramme (one grain) of thyreoid extract three times a day and also *corpus luteum* in 0.3 gramme (five grains) capsules three times a day. Food rich in cholesterolin such as grains, eggs, cream *et cetera* had been forbidden. The patient had been on this treatment for about five weeks, her general condition had improved considerably, but the xanthomatous nodules were unchanged.

THE MELBOURNE PÆDIATRIC SOCIETY.

A MEETING OF THE MELBOURNE PÆDIATRIC SOCIETY was held at the Children's Hospital on May 12, 1926, Dr. LIONEL HOOD in the chair. The meeting took the form of clinical demonstrations.

Paralytic Talipes Calcaneo-Valgus.

MR. KENT HUGHES presented a young adult patient who had suffered from *talipes calcaneo-valgus* following infantile paralysis in childhood. The deformity had been so great that the patient was forced to walk on the heel with the internal malleolus also in contact with the ground and the *os calcis* almost vertical. Mr. Kent Hughes gave an account of the various operations which have been advocated for this condition, namely those of Whitman, Dunn, Jones and Steindler and then described in detail the procedure he had followed in this case. After dividing the peroneal tendons on the lateral aspect of the foot, an incision had been made along the medial border of the sole of the foot and, the muscular layers having been retracted, the plantar fascia and the various ligaments attached to the *os calcis* had been freely divided. This had allowed the *os calcis* to be returned almost to its normal position. The operation had been performed eight months previously and the valgoid deformity was completely corrected and some power had returned in the *tendo Achillis*. The patient had a certain degree of flat foot but could walk quite well.

MR. J. G. WHITAKER remarked that he usually performed the Steindler operation for this deformity and although it was successful in most cases it was not always satisfactory. He congratulated Mr. Kent Hughes on the result obtained by the operation he had described.

Supracondylar Fracture of the Humerus.

MR. RUPERT M. DOWNES showed a boy, aged five years, who had suffered from a supracondylar fracture of the left humerus with considerable posterior displacement of the lower fragment. Several attempts at reduction had been made under general anæsthetic, but without success. A Steinmann's hook had then been inserted into the small lower fragment and extension applied by this means. The result had been very satisfactory and the elbow could be flexed completely and extended almost to its full range. Mr. Downes did not advocate this method as a routine, but recommended it for fractures which could not be satisfactorily reduced in the ordinary manner. The end result was obtained in a much shorter time.

MR. J. G. WHITAKER remarked that he had used a similar method of extension in a child with a Pott's fracture accompanied by much posterior and lateral displacement of the lower fragment. The hook had been inserted into the *os calcis* and extension applied from this point. The end result had been very satisfactory and there had been little discomfort to the patient.

MR. KENT HUGHES considered that the insertion of the hook introduced an added risk of sepsis.

MR. C. H. OSBORN considered that inserting the hook into such a small distal fragment might possibly injure the distal epiphysis of the humerus or even damage the ulnar nerve.

Encephalitis Lethargica.

DR. LIONEL HOOD showed a female patient, aged six years, suffering from a cerebral condition which had caused some difficulty in diagnosis. In January the patient had been taken suddenly ill after eating a quantity of cherries; she had had numerous convulsions, had been very irritable for one week and presented a right sided hemiparesis. This condition had persisted for some days and was accompanied by fever. The child had gradually improved, had recovered almost completely and was discharged from hospital. Two months later she had suddenly developed a similar combination of symptoms and after convulsions lasting for some hours was brought to the hospital in a comatose condition. There was no meningismus and no definite paresis, but the nervous manifestations varied continually and control of the sphincters was lost. Ophthalmoscopic examination revealed nothing abnormal and the cerebro-spinal fluid had been under increased pressure, but no abnormal cell contents had been discovered. Dr. Hood had at first considered the symptoms to be due to the so-called "cherry poisoning" or possibly to a cerebral tumour, but he now regarded the condition as *encephalitis lethargica* and he asked for any further suggestions as to

diagnosis and an expression of opinion as to the prognosis and treatment.

DR. ROBERT SOUTHEY suggested the possibility of congenital syphilis with a meningo-vascular lesion as an underlying cause.

Atypical Erythroedema.

DR. R. R. WETTENHALL showed a baby, aged sixteen months, who had manifested all the classical signs and symptoms of erythroedema including general irritability, photophobia, anorexia and sudaminal rashes and an extreme degree of atonicity, but not the typical "raw beef" hands and feet. This patient was of interest in illustrating the fact that such cases did occur and it raised the question of whether such a condition should be called "pink disease" in the absence of the pink and puffy hands and feet.

DR. LIONEL HOOD drew attention to the use of ultraviolet rays for the relief of the extreme and persistent irritability associated with the condition.

DR. J. H. KELLY said that a blood examination and leucocyte count would possibly aid in the diagnosis.

DR. D. M. EMBELTON stated that he had seen a definite improvement during the use of a mixed catarrhal vaccine in two patients.

DR. W. W. McLAREN inquired the duration of the symptoms and suggested that the hands and feet might still develop into the typical state. He had also seen some beneficial results from catarrhal vaccine.

DR. R. M. BUNTINE quoted three cases which had responded to the oral administration of horse serum.

DR. J. W. GRIEVE stated that he had seen very similar signs in patients suffering from severe and extensive lesions in infantile paralysis and suggested the possibility of an inflammatory condition of the antero-lateral columns of the spinal cord in both affections. He had also used horse serum, but without any convincing results.

DR. ROBERT SOUTHEY had also seen a baby, very like Dr. Wettenhall's patient, which had manifested irritability, photophobia, atonicity, sweating and a severe stomatitis and gingivitis, but a complete absence of the typical condition of the hands and feet.

Recurrent Synovitis of Knee Joints.

DR. D. M. EMBELTON showed a baby, aged sixteen months, who had suffered from a synovial effusion into both knee joints of some weeks' duration. The joints had not been acutely tender, but the child would not move the legs as freely as usual. At one period an urticarial rash had appeared on the chest and Dr. Embelton considered the condition was one of food sensitization and he asked for suggestions as to treatment. The X ray picture had revealed no bony changes.

DR. J. W. GRIEVE stated that he had investigated a number of children with regard to sensitization to various food substances, but without any satisfactory results. With the pollens it was different and occasionally a child was found to react definitely to the pollen of a particular flower.

DR. W. W. McLAREN quoted the case of a child who had suffered from chronic urticaria due to milk; as soon as the diet was corrected the rash had disappeared.

DR. R. R. WETTENHALL regarded the synovitis as being too persistent to be of the nature of an angioneurotic manifestation which was more commonly transient.

Cretinism.

DR. EMBELTON's second patient was a dwarfed child of three years who showed all the typical features of cretinism. The child had just come under his observation and he proposed treating it with thyroid extract.

Acidosis.

DR. EMBELTON next showed a female patient, aged six years, who had been admitted to hospital with the history of having vomited all food for the previous thirty-six hours. She had become very drowsy, was constipated for twenty-four hours and had passed no urine for twelve hours, but there was no oedema. There had not been any

jaundice. The urine had contained acetone and diacetic acid. The liver had been definitely enlarged and the bladder distended. The child had apparently been suffering from intestinal obstruction and was transferred to a surgical ward. Laparotomy had revealed a distended gall bladder and large "guinea gold" liver, but no volvulus or other cause of intestinal obstruction. The child had made an uneventful recovery. Dr. Embelton presented the patient in order to emphasize the extreme difficulty which often arose in the diagnosis between severe acidosis and acute intestinal obstruction.

Rickets.

Dr. JEAN MACNAMARA presented a boy, aged seven years, with very extreme rhachitic deformity. He was one of a large family and had lived in Fitzroy all his life. The house was one of a terrace and little sunlight reached the room where the child lived. The windows could not be opened. The diet had been very scanty and poorly balanced. She considered that the faulty feeding and lack of sunlight during early life had combined to produce severe rickets which had resulted in such gross deformity.

Mr. KENT HUGHES remarked that it was unusual to see such deformities in an Australian. It was the most severe case he had seen.

Dr. W. W. McLAREN drew attention to the fact that only the one child in a large family all on the same diet had developed rickets. He regarded the child as mentally deficient and thought this might account for the patient remaining indoors more than the others. For this reason Dr. McLaren regarded lack of sunlight as the more important factor than the dietetic in this patient.

Chronic Pyelitis.

Dr. J. W. GRIEVE showed two patients suffering from chronic pyelitis and asked for an opinion as to treatment of the condition. The first was an infant of one year who for some months had suffered from periodic attacks of fever, vomiting and irritability. The urine was loaded with pus cells and bacilli. The usual medical treatment had produced no improvement. Mr. J. T. Tait had carried out a cystoscopic examination which revealed a mild engorgement of the bladder and ureteral orifices. The ureters had been catheterized and the renal pelvis washed with silver nitrate, but the pyobacilluria still persisted.

The second child, aged two and a half years, had been operated on for an appendiceal abscess four months previously. The temperature had not subsided satisfactorily and it had been found that the urine was loaded with bacilli and pus cells. Cystoscopic examination by Mr. Downes showed some infection of the bladder mucosa. The urine from the left kidney contained pus and bacilli whilst that from the right kidney was clear. These two patients were of interest as regards the origin of the condition and its resistance to treatment.

Dr. EMBELTON regarded pyelitis as always liable to be serious owing to its possible extension. He instanced the case of a small child apparently well who was operated on for hernia and on the following day became acutely ill and very "toxic" and had died within twenty-four hours. This child had apparently suffered from a chronic pyelitis which had suddenly extended and become an acute pyelonephritis as was seen at the autopsy.

Dr. REGINALD WEBSTER stated that in the course of a number of *post mortem* examinations on children dying of pyelitis or pyobacilluria he had been impressed with the frequency with which no gross changes in the bladder or kidneys could be demonstrated. It was exceptional to see a true pyelonephritis with small points of pus under the renal capsule in these children.

Dr. H. L. STOKES said he had seen several instances of acute pyelitis with overwhelming toxic symptoms greatly improved by combined exsanguination and transfusion with whole citrated blood.

Geographical Tongue.

Dr. R. HYLTON showed a patient with a typical condition of exfoliative glossitis or so-called "geographical tongue" which had been present for some time but was not causing any gross symptoms.

Hepatomegaly.

Dr. W. W. McLAREN presented a girl, aged ten years, who had been sent down from the country on account of severe anaemia and malaise following an attack of hæmatemesis and melæna some weeks previously. The child was pale, but not jaundiced. The liver was enlarged and irregular, especially the left lobe and the spleen was mobile and easily palpable. The blood examination revealed only a secondary anaemia. There was no reaction to the Wassermann or hydatid complement fixation tests nor to the Casoni test. The corpuscular fragility was within normal limits. Dr. McLaren asked for an opinion as to the diagnosis and treatment. He regarded the condition as probably a new growth and considered laparotomy was indicated.

Dr. G. PENINGTON suggested Banti's disease as a possible diagnosis and advocated splenectomy as a means of removing a focus of infection.

Dr. HYLTON stated that the child had been acutely ill on admission to hospital and was then suffering from severe oral sepsis. Since the mouth had improved, the patient's general condition was very much better.

Dr. R. M. BUNTINE quoted a similar patient with enlarged liver and spleen associated with a condition of chronic septicæmia.

Dr. J. W. GRIEVE had seen a similar clinical picture produced by a condition of irregular cirrhosis of the liver, but this was more likely to be associated with a certain degree of jaundice.

Pathological Specimens.

Dr. REGINALD WEBSTER demonstrated an excellent collection of specimens. The first was an example of the unusual condition of pseudomyxoma of the peritoneum arising from the appendix. This specimen was from an adult patient and was of extreme interest on account of its close resemblance to colloid carcinoma (see page 703).

The next specimen was a colour preparation of an acutely inflamed appendix which contained over one hundred thread worms.

Then followed a number of viscera from a patient who died of a chronic septicæmia of four months' duration. The *Staphylococcus aureus* had been isolated from a blood culture *ante mortem* and from lesions in the various viscera. The liver was enlarged and the site of a unilobular cirrhosis, the spleen and kidneys presented septic infarcts.

Another specimen of considerable interest was the lung of a child aged three years containing a large well defined tuberculous cavity at the upper lobe and a massive bronchopneumonia involving the remainder of the viscus. This child had recently emigrated to Australia and had been extremely ill on arrival in Melbourne. Tubercle bacilli had been present in the sputum in large numbers before death.

The last specimen was a segment of small intestine with typical typhoid ulcers which had become peculiarly stained almost black with bile, the latter producing a most beautiful natural colour preparation.

Correspondence.

FRACTURE OF THE NECK OF THE FEMUR.

SIR: In a recent issue of the journal (May 1) Dr. M. L. Scott contributes a paper on the treatment of fractures of the lower extremity. In a section on fracture of the neck of the femur he recommends a method of treatment in a Thomas splint with extension and abduction. Then he says: "Treated in this way, these fractures even in elderly folk almost always will unite. The impression still seems to prevail that fractures of the neck of the femur do not usually unite. Such teaching has had a pernicious influence. If we aim at union, we get it."

This is something we ought to hear more about. The statement is not qualified and it must be taken to apply to the common form of fracture of the neck of the femur, the intracapsular. I have seen and treated a very large number of fractures of the femur, over two hundred in the past ten years and among them a number of intracapsular fractures of the neck, but of the latter kind I have seen only one case in a child. In a number of them, including this child, I have aimed at union by extension and abduction in a Hodgen splint and some have been treated in plaster of Paris. I have taken a great deal of trouble over them and have carried out the treatment as well and carefully as I knew how.

My marksmanship does not seem as good as Dr. Scott's, because when I have aimed at union I have invariably missed. There is not a single success to record in the whole lot.

Now I confess to teaching what Dr. Scott declares "has had a pernicious influence." I have this much justification, however, that I teach it from personal experience.

If there is a secret of success, I do not hold it. Dr. Scott claims that he holds it. So, apparently, do some American writers, but they are a long way off. I venture to say that in a matter of such importance it would oblige many of us and teach us a great deal (it might reverse my teaching) if Dr. Scott could see his way to publish some of the skiagrams of his successful cases, especially of those old people who are usually the subjects of this injury. And I would like details and especially follow-up details.

I am sincerely anxious to teach students only what is right and sound. I believe I have an open mind, though I admit it is a somewhat sceptical one—one has to be cautious. I want to be quite sure. Am I really teaching pernicious stuff?

Yours, etc.,

C. E. CORLETTE.

175, Macquarie Street, Sydney,
May 22, 1926.

THE PROFESSION AND THE STATE.

SIR: In your leader on the above subject (May 22, 1926) in regard to the report of the Royal Commission on Health, one reads: "Weeks and months have passed; the members of the Branches of the British Medical Association in Australia have not manifested any real interest in the matter."

As you say, this lack of interest in such an important matter is to be regretted and should be of grave concern to the Federal Committee.

An independent observer reading your leader might ask: "Have the members of the British Medical Association in Australia any interest in the Association?"

Is not the time now opportune for the Federal Committee to consider a question such as this?

A distinguished member of the profession, recently in our midst, said: "Out here, of course, the B.M.A. is 'the thing.'" The inference apparently was that in Britain there are other bodies that mean more to many practitioners than the British Medical Association. Are we approaching the time where the same sort of thing is about to happen in Australia? If so, such a happening is to be deeply deplored.

Correspondence in THE MEDICAL JOURNAL OF AUSTRALIA seems to indicate that all is not well with the profession. Prominent members make charges of dishonesty and incompetence on the part of certain of their professional brethren.

It is undoubtedly right that the public should be protected, but on the other hand it is right that the practitioner should be able to make a reasonable living by legitimate means. If the charges made against certain practitioners are true, it is because they are forced into certain positions owing to the necessity of having to make a living. Charging excessive fees, undertaking of special work by the unqualified, fee-splitting *et cetera* are all practices that tend to become common when competition is too keen.

The time has come when the Federal Committee should earnestly consider these matters. If practitioners have to descend to such meanness it is unlikely that they will be useful members of the British Medical Association.

Another source of weakness in the Association is the men who are in an assured position and are non-cooperators in the Association's work. They should have the time and energy to stand by the officers of the Association and do all in their power to assist the man on a small income and the man who is having a hard time at the hands of a country hospital committee.

Unless those in authority—and one naturally thinks of the Federal Committee—take some steps to overcome this non-cooperation, the time is not far off when the British Medical Association will not be "the thing" in Australia.

A little more unselfishness on the part of the well-to-do in the profession would go a long way to strengthen the Association. Passing resolutions in capital cities is not enough. If one prominent man in the Association traversed each State once in two years to study the details of conditions of medical practice, it would be greatly to the advantage of the profession and the State.

If there is discontent and non-cooperation on the part of the profession from any cause whatever, the ideals of the Federal Commission on Health will be realized in very small measure indeed, as so much depends on the cooperation of practitioners generally.

It is on the leadership and broadness of vision on the part of the Federal Committee that the future of the British Medical Association in Australia depends and leadership and broadness of vision are needed more than ever before.

Yours, etc.,

"OFFICER."

June 4, 1926.

WHAT IS SURGERY?

SIR: In a review of an article with the above title by E. A. Graham, the following appears: "The Medical Schools affiliated with the University Hospitals should be temples of learning in which specially gifted young men ambitious to become surgeons will be given maximal opportunities to prepare their minds and develop their manual skill simultaneously."

"One of the chief aims in the training of surgeons is the development for tomorrow of surgeons who will be better than those today. Adequately equipped laboratories for experimental work on animals and in chemistry, bacteriology and pathology are essential."

How long will it be before this state of affairs exists in Australia? Dr. R. Gordon Craig has set an example that might be followed by many and has pointed the way in no uncertain manner to the "powers that be." The profession and the public are greatly in his debt.

Yours, etc.,

HONORARY SURGEON.

June 4, 1926.

RADIATION IN THE TREATMENT OF PEPTIC ULCERS.

SIR: The medical and surgical failures in the treatment of peptic ulceration mentioned by Dr. Pitcher in his paper in your journal of May 22, 1926, are very interesting. The radio-diagnosis has been correlated with the clinical diagnosis in the approved manner, but it would have been beneficial to those failures if radiotherapy had been correlated with ordinary therapy.

Alkalinization need not be pushed to the danger of alkalosis, if the hyperacidity is corrected by short wave X radiation which at the same time will relieve the spasm and even cause healing of the ulcer, provided that the

¹ This review did not appear in THE MEDICAL JOURNAL OF AUSTRALIA.—Editor.

surrounding induration does not form a mechanical bar to the healing process.

Cases of recurring or relapsing ulceration as Case I. (page 572) are frequently associated with lowered calcium metabolism and while this persists, they remain medical and surgical failures. Ulcers are hard to heal in any situation while the patient exhibits calcium deficiency. The administration of parathyroid extract will often convert a deficient calcium metabolism into a normal one, but not so readily (though it operated well in the case reported) as will radiation with the much longer waves of the ultraviolet light spectrum.

This radiation improves the calcium metabolism (compare its use in rickets) and so allows healing to occur. Your correspondent (E. Mackenzie) in the same issue pays a tribute to the ulcer healing properties of this radiation, though in his case it is applied as direct sunlight to ulcers on the leg. In all of these ulcer cases the action of the ultraviolet light is the same in the restoration of the calcium metabolism to normal.

Dr. Pitcher will find both these forms of electromagnetic radiation valuable additions to his therapeutic equipment in ulcer cases. I hope he will at a subsequent date report his findings after treating his peptic ulcer cases by deep therapy to the stomach (Holzknecht and Ledoux Lebard) and ultraviolet light to the body generally.

Yours, etc.,

BEDE J. HARRISON.

235, Macquarie Street, Sydney.
May 25, 1926.

Obituary.

EDGAR MONTGOMERY INGLIS.

THE death of Dr. Edgar Montgomery Inglis which occurred at Kew, Victoria, on April 26, 1926, has removed from the ranks of the medical profession one who had given many years of faithful service to the community, one who was held in honour by his colleagues and one whose example might well be followed by many of the younger generation.

Edgar Montgomery Inglis was born at Coburg, Victoria, on June 16, 1856. He was educated at Wesley College. From this school he passed his matriculation examination and, having chosen medicine as his life's work, he journeyed to Scotland and became an undergraduate at the University of Edinburgh. He graduated as bachelor of medicine and master of surgery in 1886. He also obtained the diplomas of licentiate of the Royal College of Physicians, of the Royal College of Surgeons of Edinburgh and of the Faculty of Physicians and Surgeons of Glasgow. Immediately after graduation he went to Dublin and took a special course in obstetrics at the Rotunda Hospital. This stood him in good stead. In later years when obstetrics formed a large part of his practice, he used the Rotunda methods.

Towards the end of 1886 Edgar Montgomery Inglis returned to Australia. He took up practice in Kew, Victoria, and remained there in active work till the time of his death. He was a diligent worker and his practice naturally became extensive. His chief pride was in his midwifery work. He was Medical Officer of Health to the City of Kew and held the position for a continuous period of thirty-two years. His only other public interest lay in the Trinity Grammar School, Kew. He was one of the founders of this institution and acted as its treasurer for many years. He was a member of the school council until the time of his death.

In his younger days Edgar Montgomery Inglis was an enthusiastic tennis player and cyclist. During his university days he was awarded a half-blue for his proficiency in both these branches of sport. With the passing of the years he took up the less strenuous pastimes of shooting and fishing. He was particularly fond of these recreations. In the later years of his life his interests were confined

to his garden. Unfortunately his holidays were relatively infrequent and this doubtless had an injurious effect on his health. He married Miss A. J. Anderson, of Jedburgh, whom he met in Scotland. Mrs. Inglis and five sons survive him. Two sons are members of the medical profession and one is carrying on his father's practice. They can look back with thankfulness to the life of one who lived and worked for the benefit of his fellows.

FRANK CARL FREDERIC ANDREW.

WE regret to announce the death of Dr. Frank Carl Frederic Andrew which occurred at Melbourne on April 7, 1926.

Proceedings of the Australian Medical Boards.

NEW SOUTH WALES.

THE undermentioned have been registered, under the provisions of *The Medical Act, 1912 and 1915*, as duly qualified medical practitioners:

- Crowley, Vivian George, L.M.S.S.A., 1926 (London), 16, Drummoyne Avenue, Drummoyne.
Godsall, John Rex, M.B., Ch.M., 1926 (Univ. Sydney), Pomeroy, Macleay Street, Potts Point.
Green, Alan Kenneth, M.B., Ch.M., 1926 (Univ. Sydney), Kenwyn, Percy Street, Warwick, Queensland.
Lieberman, Hyman Barnett, M.B., Ch.M., 1926 (Univ. Sydney), 41, Darley Road, Randwick.
Manoy, Augusta, M.B., Bac. Surg., 1922 (Univ. New Zealand), Duntroon Avenue, Roseville.
Mills, Dorothy Isabel, M.B., Ch.M., 1926 (Univ. Sydney), Fox Valley Road, Wahroonga.
Nothling, Otto Ernest, M.B., 1926 (Univ. Sydney).
Paterson, Robert Francis, M.B., Ch.M., 1926 (Univ. Sydney), Melbourne, 69, Middle Head Road, Mosman.
Wallace, Kenneth Stewart, M.B., Ch.M., 1926 (Univ. Sydney), 15, Shell Cove Road, Cremorne.

For Additional Registration.

- Barnes, Jack, Ch.M., 1926 (Univ. Sydney).
Cunningham, Archibald James, M.D., 1926 (Univ. Sydney), 42, Susan Street, Randwick.
Metcalfe, Arthur John, D.P.H., 1926 (Univ. Sydney), Russell Street, Vaucluse.
Stobo, Alexander Jarvie Hood, Ch.M., 1923 (Univ. Sydney), M.R.C.P., 1926 (Lond.).
Tooth, Frederick, D.P.H., 1925; R.C.P.S. (Eng.).

VICTORIA.

THE undermentioned has been registered, under the provisions of Part I. of the *Medical Act 1915*, as a duly qualified medical practitioner:

- Weaver, Ralph Edward, M.B., Ch.M., 1920 (Univ. Sydney), Eye and Ear Hospital, Melbourne.

Additional Diplomas Registered.

- Fairley, Neil Hamilton, M.D., 1917 (Univ. Melbourne); D.T.M. & H., 1920 (Camb.); M.R.C.P., 1920 (Lond.).
Fairley, Keith Douglas, M.R.C.P., 1924 (Lond.).
Hall, Reginald Dalton McKellar, F.R.C.S., 1925 (Edin.).

QUEENSLAND.

THE undermentioned have been registered, under the provisions of *The Medical Act of 1925*, as duly qualified medical practitioners:

- Backwell, Claude Ewart, M.B., B.S., 1922 (Univ. Melbourne), Caboolture.
 Cherry, George Frederick, M.B., B.S., 1919 (Univ. Melbourne), Brisbane.
 Crooks, Arthur Augustus, M.B., B.S., 1913 (Univ. Melbourne), Townsville.
 Gibson, Leslie Wylie Norman, M.B., Ch.M., 1926 (Univ. Sydney), Brisbane.
 Hayes, Geoffrey Stanhope Sautelle, M.B., Ch.M., 1926 (Univ. Sydney), Toowoomba.
 Hooper, Ivan Gregory, M.B., Ch.M., 1926 (Univ. Sydney), Toowoomba.
 Jamieson, George Arthur, M.B., B.S., 1925 (Univ. Melbourne), Herberton.
 Law, Thomas Boyd, M.B., Ch.M., 1924 (Univ. Sydney), Maleny.
 Watkins, Eric Roderick, M.B., 1926 (Univ. Sydney), South Brisbane.
 Uhr, Clive Wentworth, M.B., Ch.M., 1926 (Univ. Sydney), Gatton.
 Dodson, George Hirst, M.B., Ch.M., 1921 (Univ. Sydney), Brisbane.
 Douglas, William Joseph Fletcher, M.B., Ch.M., 1925 (Univ. Sydney), Brisbane.
 Elliot-Smith, Mervyn Harrie, M.B., Ch.M., 1923 (Univ. Sydney), Brisbane.
 Fava, Amadee, M.D., 1922 (Univ. Malta), Brisbane.
 Higgins, Joseph Vincent, M.B., B.S., 1925 (Univ. Melbourne), Toowoomba.
 Roberts, Herbert Spencer, M.B., Ch.M., 1925 (Univ. Sydney), Maryborough.
 Johnson, Horace William, M.B., Ch.M., 1925 (Univ. Sydney), Brisbane.
 Power, Harold Horne, M.B., Ch.M., 1925 (Univ. Sydney), Brisbane.
 Lee, Alan Edward, M.B., B.S., 1920, M.D., 1922 (Univ. Melbourne), F.R.C.S., 1924 (Eng.), Brisbane.
 Grant, Robert, M.B., Ch.B., 1923, F.R.C.S., 1925 (Edin.), Mackay.
 Kirkland, William Bruce, M.B., B.S., 1925 (Univ. Melbourne), Charleville.
 O'Donnell, Kenneth Francis, M.B., B.S., 1924 (Univ. Melbourne), Mackay.
 Ponton, Ronald George, M.B., Ch.M., 1923 (Univ. Sydney), Virginia.

Additional Qualifications.

- Elliot-Smith, Mervyn Harrie, F.R.C.S., 1925 (Edin.).
 Murray, Gerald Aubrey, D.P.H., 1925 (Univ. Sydney).

TASMANIA.

THE undermentioned have been registered, under the provisions of *The Medical Act 1918*, as duly qualified medical practitioners:

- Romeo, Giuseppe, M.D., Ch.D., 1894 (Naples), Penguin.
 Howson, Frank, M.R.C.S., L.R.C.P. 1910 (London), Swansea.
 Drake, Francis James Bain, M.B., B.S., 1922 (Univ. Melbourne), Launceston.

RECIPROCITY BETWEEN THE UNITED KINGDOM AND ITALY.

WE are indebted to the Medical Board of Victoria for the following information. On May 21, 1925, an agreement was signed at Rome governing the conditions "to regulate the professional practice of medical practitioners" in the United Kingdom and Italy. Clause I. of the agreement affects the rights and privileges of Australian practitioners and of Italian practitioners desiring to practise in Australia. It reads as follows:

Medical practitioners holding diplomas issued by licensing bodies in Great Britain, in the British colonies, in India and in the British possessions and

Dominions between which and Great Britain reciprocity exists, and being legally entitled through existing legislation and on the basis of their diplomas and the inscription thereon in the Medical Register in Great Britain to carry on free professional practice in these territories, can be inscribed on the professional registers of the *Ordine dei Medici* of the Kingdom of Italy . . . and pursue accordingly their professional practice in the Kingdom of Italy and its colonies without the need of undergoing any further examinations or obtaining any new qualification in the institutes of the Kingdom of Italy.

This clause will consequently have application to Australia, even in those States of which the *Medical Act* does not contain a "reciprocity clause."

THE MEDICAL DIRECTORY.

CIRCULARS containing forms to be filled in for the compilation of the Medical Directory have been addressed to every medical practitioner in Australasia. Those who did not reply to the first invitation, received a second circular. There are still many who have not entered the details required on the forms and returned them to The Printing House, Seamer Street, Glebe, New South Wales. The form has been reproduced in this issue and will be found in the Advertiser. Members who have not already sent in their forms, are earnestly requested to tear out the page, to fill in the necessary details and to transmit it to this address as soon as possible.

LISTS OF MEMBERS.

INFORMATION has been received from the Honorary Secretaries of the Branches of the British Medical Association concerning a few further omissions of the indication of war service in the lists of members published as a supplement to THE MEDICAL JOURNAL OF AUSTRALIA of February 13, 1926. The Honorary Secretaries of the Branches depend on information supplied to them by the members for accuracy of these lists. Members should therefore notify the Honorary Secretaries if any omissions or inaccuracies are detected.

The following entries should be substituted for those which have appeared:

- X Barbour, W. M., Gunning, New South Wales.
 X Maunder, H. A., D.S.O., Melbourne Hospital, Victoria.
 X Nicholson, L. F., 125, Grange Road, Glen Huntly, Victoria.
 X Ward, W. H., 600, Barkly Street, Footscray, Victoria.

Medical Appointments.

Dr. Kenneth Francis O'Donnell (B.M.A.) has been appointed Acting Government Medical Officer at Mackay and Acting Visiting Surgeon to His Majesty's Gaol, Mackay, Queensland.

Dr. John Coffey (B.M.A.) has been appointed Deputy Commissioner of Public Health, Queensland.

Dr. Reginald Williams has been appointed Visiting Medical Officer to the Aboriginal Settlement at Taroom, Queensland.

Dr. Paul Ernest Voss (B.M.A.) has been appointed Acting Government Medical Officer at Rockhampton and a Health Officer under *The Health Acts*, 1900 to 1922, Queensland.

Dr. Cecil Evelyn Cook (B.M.A.) has been appointed a Health Officer, Home Secretary's Department, Brisbane.

Dr. James Bogle (B.M.A.) has been appointed Government Medical Officer at Clermont, Queensland.

Dr. George William Macartney (B.M.A.) has been appointed Acting Medical Officer at Brisbane, St. Helena Penal Establishment and Brisbane Prison and Acting Medical Officer to the Diamantina Orphanage, Brisbane.

Dr. Thomas Robert Quinn (B.M.A.) has been appointed Acting Visiting Medical Officer to the Westwood Sanatorium, Queensland.

The undermentioned have been appointed to the Commission of the Peace for the State of New South Wales: Dr. John Bostock (B.M.A.), Dr. Clifford Henry (B.M.A.) and Dr. Eric Theodore Hilliard (B.M.A.).

Dr. Walter Lockhart Gibson (B.M.A.) has been appointed a Resident Surgeon at the Royal Westminster Ophthalmic Hospital, London, as from May 1, 1926. Dr. Gibson has until recently held the position of acting Resident Surgeon at the Royal London Ophthalmic Hospital, Moorfields, London.

Dr. Murray Sanderson has been appointed District Medical Officer and Public Vaccinator at Cue, Western Australia.

Dr. Ralph Alderton Baker (B.M.A.) has been appointed Honorary Medical Officer at the Barmera Hospital, South Australia.

Dr. Selwyn Bligh Sutton (B.M.A.) has been appointed Medical Officer at the Barmera Hospital, South Australia.

Dr. Gerald J. Stoney (B.M.A.) has been appointed Officer of Health to the Board of Health in the district of Hammond, South Australia.

Dr. W. P. White (B.M.A.) has been appointed District Medical Officer and Public Vaccinator, Wickiepin, Western Australia.

Dr. Claude Vincent Hallett (B.M.A.) has been appointed Medical Inspector of Seamen at the port of Thevenard, South Australia.

Books Received.

MODERN METHODS IN THE DIAGNOSIS AND TREATMENT OF HEART DISEASE, by Francis Heatherley, M.B., B.S. (London), F.R.C.S.; Second Edition; 1926. London: Baillière, Tindall and Cox. Post 8vo., pp. 282. Price: 8s. 6d. net.

MODERN MEDICINE, ITS THEORY AND PRACTICE, edited by Sir William Osler, Bart., M.D., F.R.S., Re-edited by Thomas McCrae, M.D., Assisted by Elmer H. Funk, M.D.; Volume II.: Diseases of Doubtful Etiology *et cetera*; 1925. Philadelphia: Lea and Febiger; Sydney: Angus and Robertson, Limited. Royal 8vo., pp. 901, with illustrations. Price: 42s. net.

RELIGIOSITY AND MORBID MENTAL STATES, by H. I. Schae, M.D.; Translated from the Danish by W. Worster, M.A.; 1926. London: Methuen and Company, Limited. Post 8vo., pp. 120. Price: 5s. net.

THE MEDICAL ANNUAL: A YEAR BOOK OF TREATMENT AND PRACTITIONER'S INDEX; 1926. Bristol: John Wright and Sons, Limited. Royal 8vo., pp. 616, with illustrations. Price: 20s. net.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xxii.

THE ADELAIDE CHILDREN'S HOSPITAL, INCORPORATED: Two Resident Medical Officers.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C. 1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phoenix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association.
QUEENSLAND: Honorary Secretary B.M.A. Building, Adelaide Street, Brisbane.	Brisbane United Friendly Society Institute. Stannary Hills Hospital. Cook District Hospital.
SOUTH AUSTRALIAN: Honorary Secretary, 12, North Terrace, Adelaide.	Contract Practice Appointments at Ceduna, Wudinna (Central Eyre's Peninsula), Murat Bay and other West Coast of South Australia Districts.
WESTERN AUSTRALIAN: Honorary Secretary, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Diary for the Month.

- JUNE 21.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JUNE 22.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 22.—Illawarra Suburbs Medical Association, New South Wales.
JUNE 23.—Victorian Branch, B.M.A.: Council.
JUNE 23.—Western Medical Association (Forbes), New South Wales.
JUNE 24.—New South Wales Branch, B.M.A.: Branch.
JUNE 25.—Queensland Branch, B.M.A.: Council.
JULY 1.—South Australian Branch, B.M.A.: Council.
JULY 2.—Queensland Branch, B.M.A.: Branch.
JULY 6.—New South Wales Branch, B.M.A.: Council (Quarterly).
JULY 6.—Tasmanian Branch, B.M.A.: Council.
JULY 7.—Victorian Branch, B.M.A.: Branch.
JULY 7.—Western Australian Branch, B.M.A.: Council.
JULY 8.—New South Wales Branch, B.M.A.: Clinical Meeting.
JULY 8.—Victorian Branch, B.M.A.: Council.
JULY 9.—Queensland Branch, B.M.A.: Council.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.